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AMERICAN ACADEMY
of
OPHTHALMOLOGY
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JULY - AUGUST
1960

VOLUME 64 NUMBER 4

ANNUAL MEETING, OCTOBER 9-14, 1960, CHICAGO

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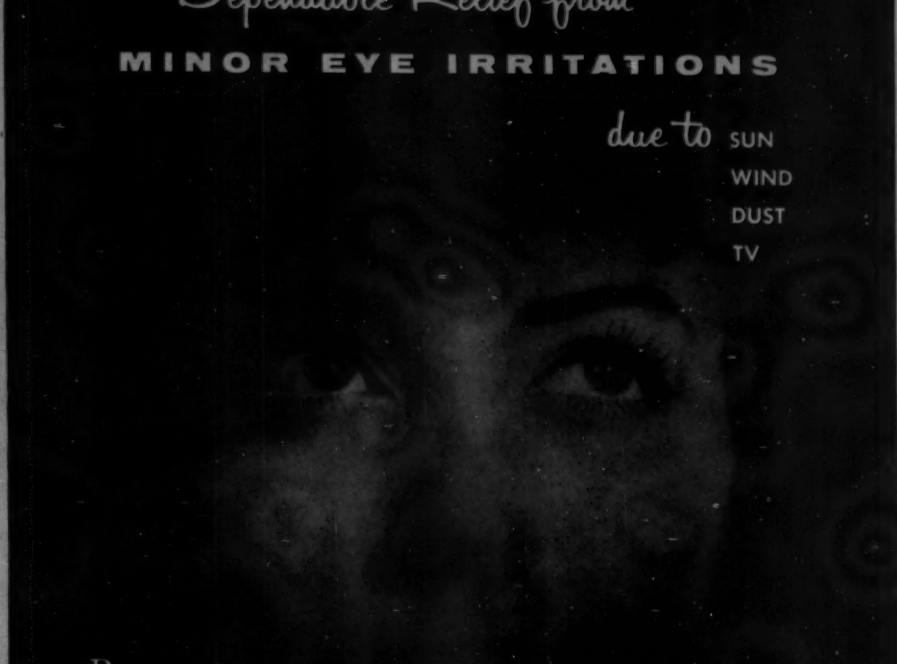

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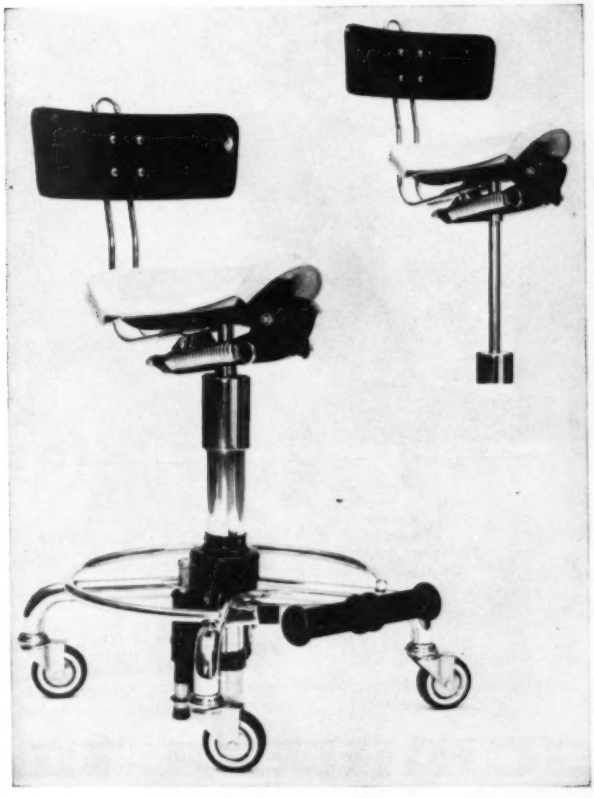

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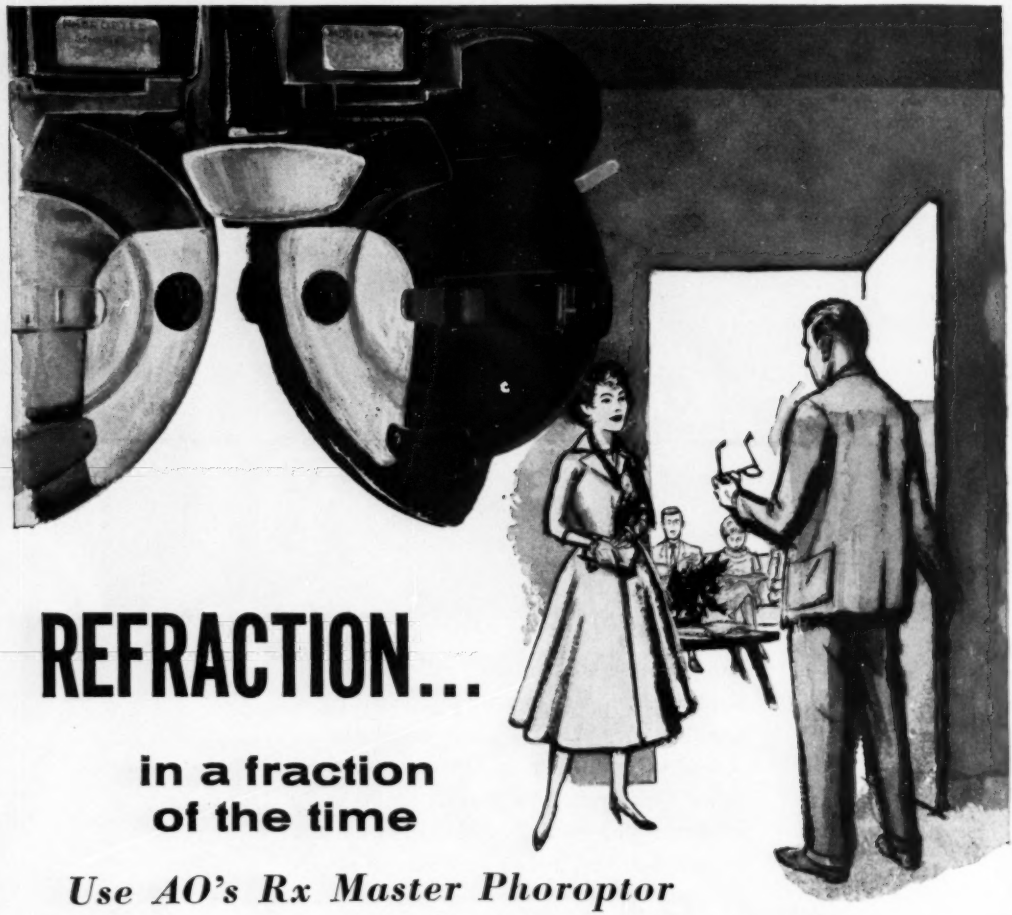

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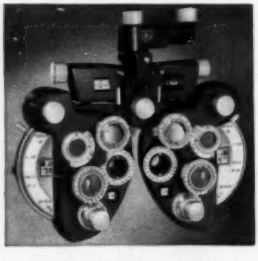

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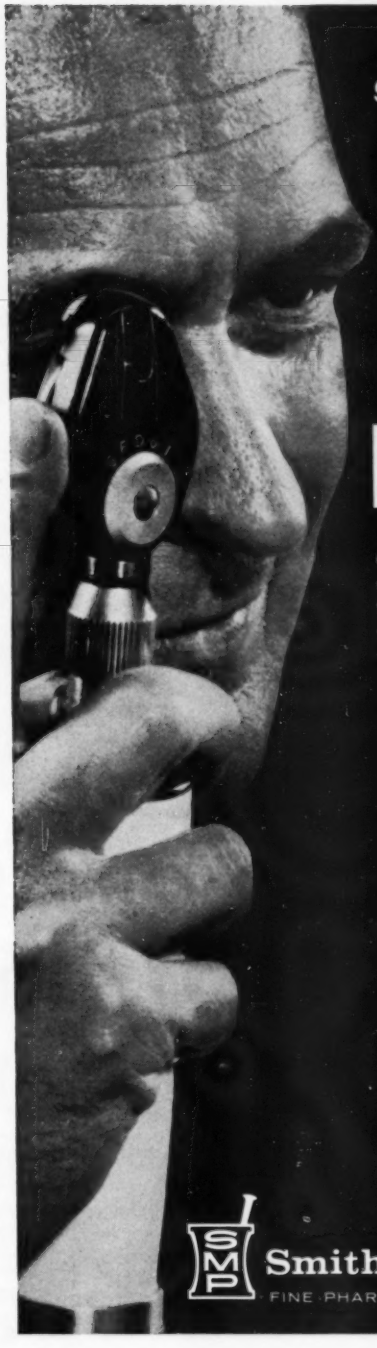

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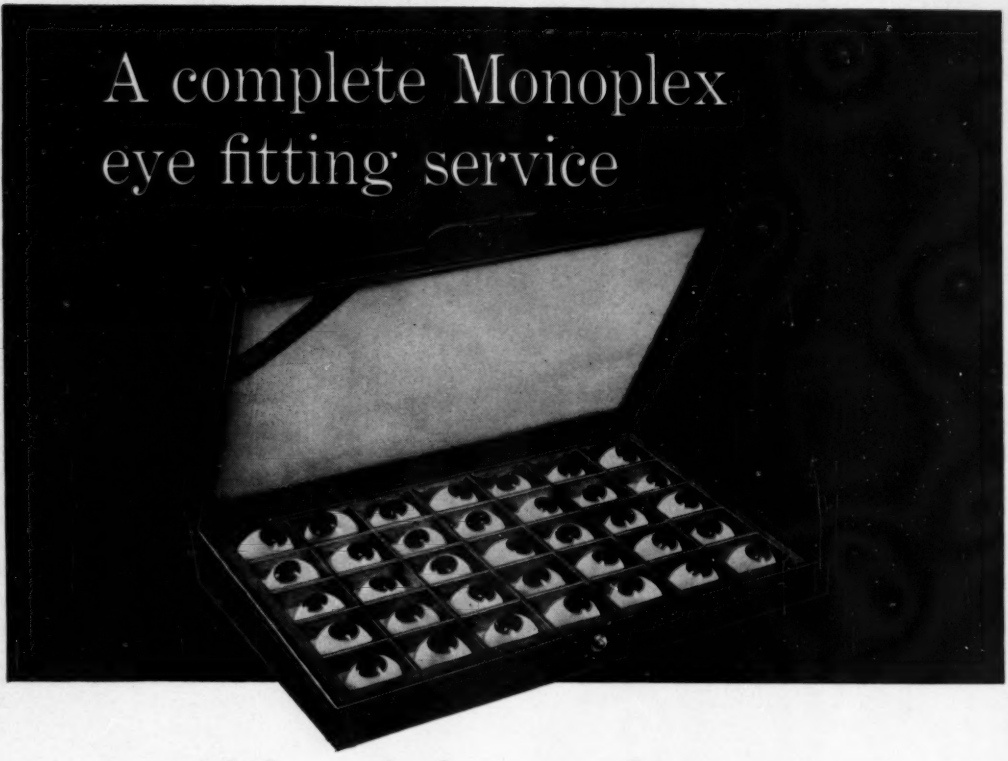

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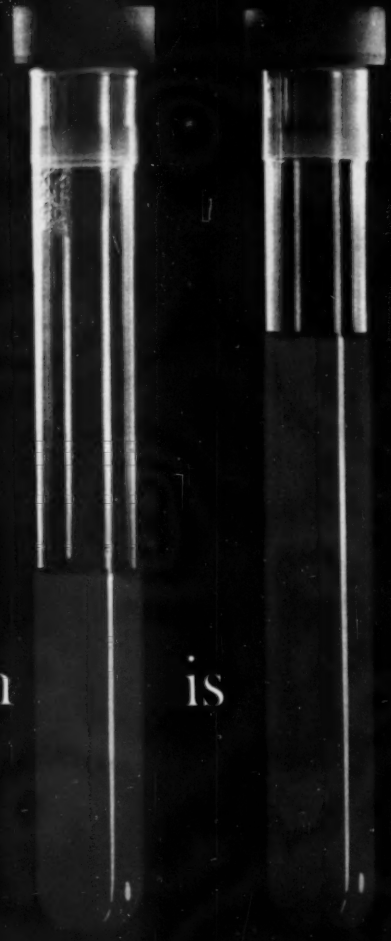

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2. Cherashin, E.: J. Am. Dent. Assn., 58:17 (Apr., 1959).

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1. Derouaux, G., and Roskam, J.: Adrenaline, Adrenalone, and Mean Bleeding Time, J. Physiol. 90:65 (1937).
2. Roskam, J.: Arrest of Bleeding, Charles C Thomas, Springfield, Ill. (1954).
3. Peele, J.C.: Adrenosem in the Control of Hemorrhage from the Nose and Throat, A.M.A. Arch. of Otolaryng. 61:450 (Apr., 1955).
4. Fulton, G.P., Lutz, B.R., Shulman, M.H., and Arenot, K.A.: Moccasin Venom as a Test for Susceptibility to Petechial Formation in the Hamster. Venoms: Published by the Am. Assoc. for the Advancement of Science (1954).
5. Pappenheimer, J.R.: Passage of Molecules Through Capillary Walls, Physiol. Rev. 33:387 (July, 1953).
6. Escala, J.C.: The Use of the Systemic Hemostat Carbazochrome Salicylate, West. J. Surg., Obstet., and Gynec. 64:88 (Jan., 1956).
7. Kingsbury, B.C., Jr., and Young, H.E.: A Preliminary Report on Adrenosem Salicylate for Control of Hemorrhage, J. Calif. State Dental Assn. and Nev. State Dental Assn. 31:163 (May-June, 1955).
8. Moss, A.A.: Control of Hemorrhage in Dental Surgery, Dental Survey 32:1622 (Dec., 1956).
9. Ryan, Allan J.: Control of Bleeding in Familial Telangiectasia, The Meriden Hosp. Bull. 7: (Jan., 1958).
10. Peele, J.C.: Further Observations on the Use of Adrenosem Salicylate in the Control of Hemorrhage from the Nose and Throat, N. Car. Med. J. 17:98 (Mar., 1956).
11. Peele, J.C.: Control of Hemorrhage from the Nose and Throat, Med. Times 86:1228 (Oct., 1958).
12. Brode, H.A., and Chianese, T.C.: A Clinical Evaluation of Adrenosem Salicylate, Ann. of Dentistry 15:56 (Sept., 1956).
13. Owings, Capers B.: The Control of Postoperative Bleeding with Adrenosem, Laryngoscope 65:21 (Jan., 1956).
14. Perkins, R.E.L.: A Clinical Investigation of Adrenochrome Monosemicarbazone - Sodium Salicylate, Oral Surg., Oral Med., Oral Path. 10:230 (Mar., 1957).
15. Zubiate, C.B., and Escanaverino, R.S.: Adrenosem for the Prevention of Bleeding in Tonsillectomy, Am. Pract. & Dig. of Treatment 8:365 (Mar., 1957).
16. Orzac, E.: Medical Care of the Child Patient Before and After Adenoidectomy and Tonsillectomy, N.Y. State J. Med. 56:886 (Mar., 1956).
17. Riddle, A.C., Jr.: Adrenosem Salicylate: A Systemic Hemostatic, Oral Surg., Oral Med., Oral Path. 8:617 (June, 1955).
18. Roberts, E.W.: Observations on the Use of Adrenochrome in Dental Practice, Oral Surg., Oral Med., Oral Path. 10:52 (Jan, 1957).
19. Brown, W.S.: Control of Bleeding After Dermabrasion, Northwest Med. 57:470 (Apr., 1958).
20. Dimmette, J.E., and Terry, J.G.: Better Control of Bleeding in Vaginal Hysterectomy, presented before the Texas State Med. Society, Surgical Section (Jan., 1956).
21. Sherber, D.A.: The Control of Bleeding, Am. J. Surg. 86:331 (Sept., 1953).
22. Dempsey, P.J.: The Care of the Prostatic Cavity, S. African Med. J. 30:381 (Apr., 1956).
23. Ersner, M.S., and Lerner, S.S.: The Unsolved Problem of the Tonsils and Adenoids, The Med. Clinics of N. Amer. 40:1749 (Nov., 1956).
24. Real, H.B.: Minimizing Postoperative Ecchymosis, The Am. J. of Surgery 96:751 (Dec., 1958).
25. Coyte, J.E.: Analysis of Blood and Vascular Factors in the Prophylaxis of Tonsillo-Adenoidal Hemorrhage, Laryngoscope 10:1029 (Oct., 1957).
26. Proctor, D.F., and Douglass, C.C.: Bleeding Following Tonsil and Adenoid Operations, Trans. Amer. Acad. Ophthal. Otolaryngol. 62:592 (July-Aug., 1958).
27. Georgoyne, J.R.: Adrenosem and the Bleeder (in press)
28. Nicely, P., F.A.C.S., F.I.C.S. (Personal communication).
29. Wilkins, S.D.: Gastrointestinal Bleeding as Seen by the Proctologist, J.A.M.A. 163:1214 (April. 6, 1957).
30. Feinblatt, T.H., Feinblatt, H.M., and Ferguson, E.A.: Successful Non-Surgical Uses of Carbazochrome Salicylate, Amer. Pract. & Dig. of Treatment, 9:1827 (Nov., 1958).
31. Stich, M.H.: Carbazochrome Salicylate Therapy in Hereditary Hemorrhagic Telangiectasia, N.Y. State J. of Med., 59:2725 (July 15, 1959).

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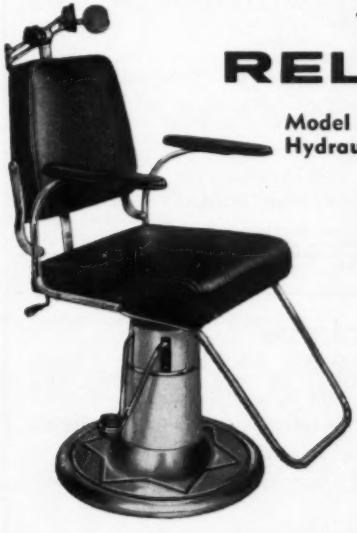

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2. *M. Times* 84:741, 1956.
3. *Am. J. Ophth.* 42:771, 1956.
4. *Southwestern Med.* 40:120, 1959.

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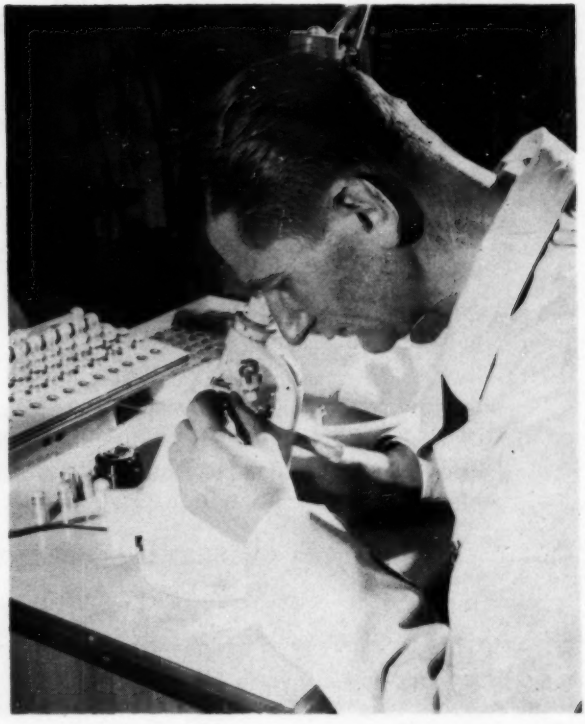

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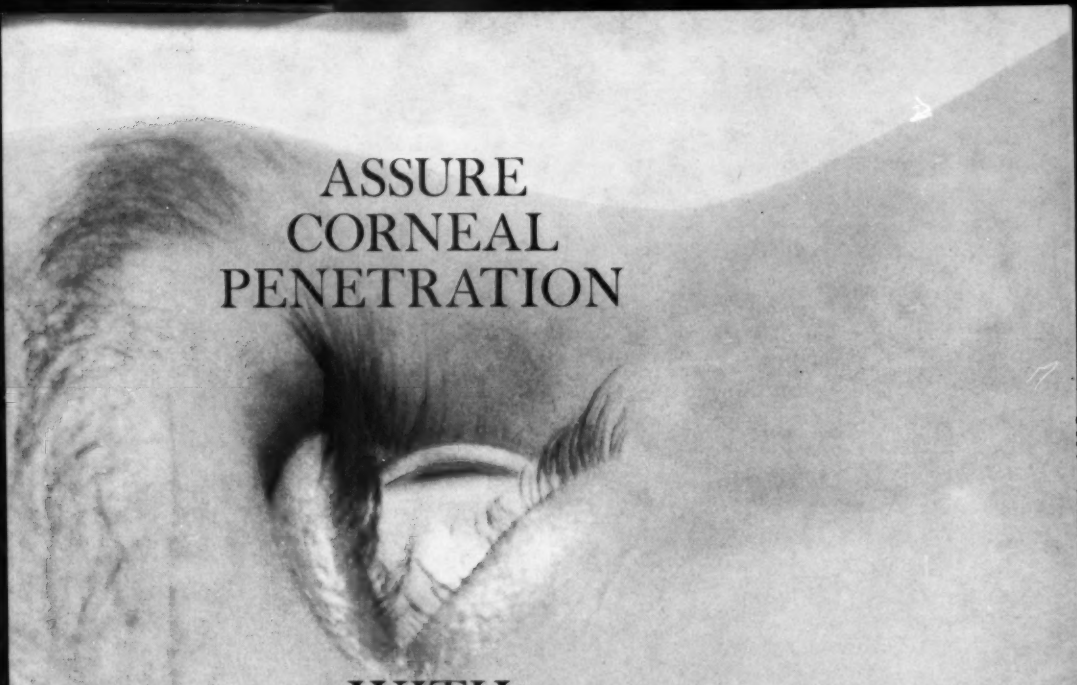

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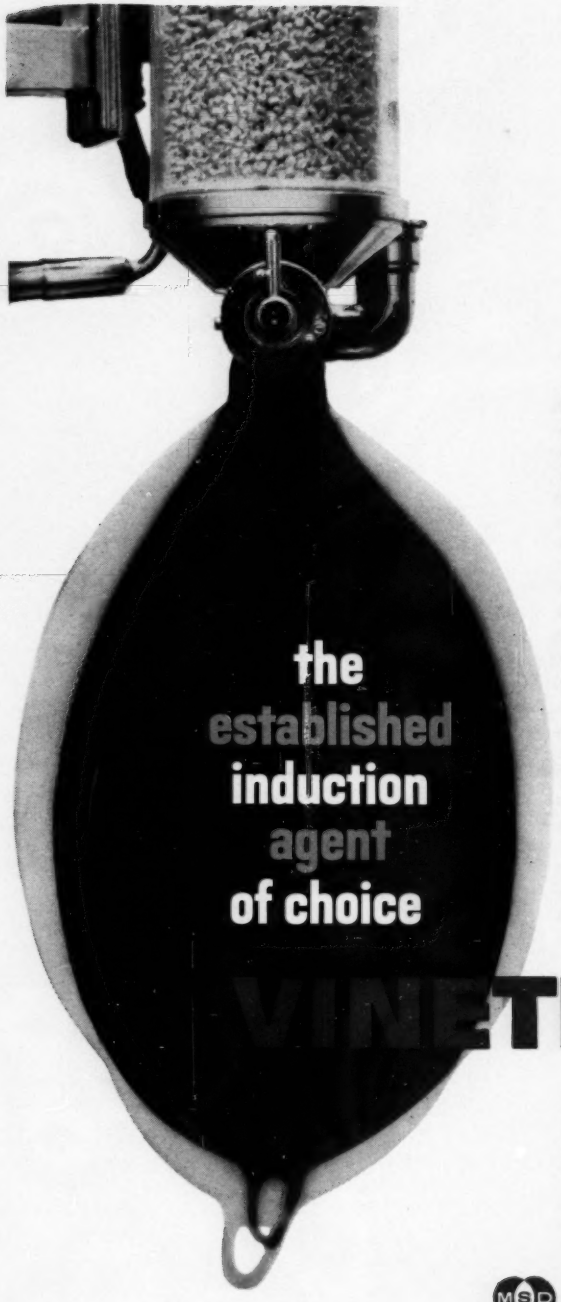

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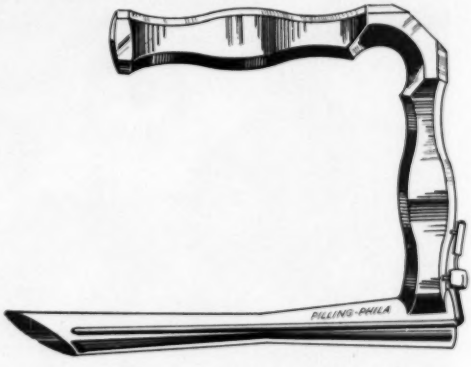

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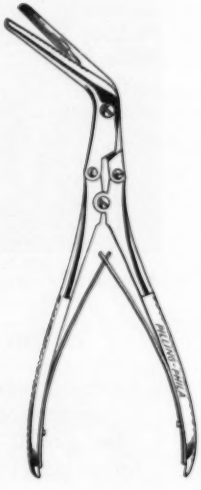

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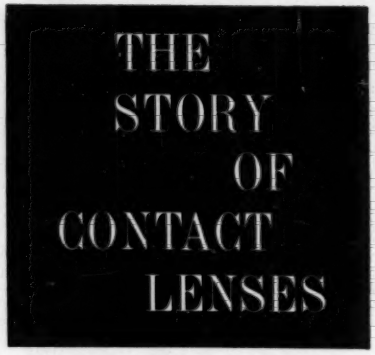

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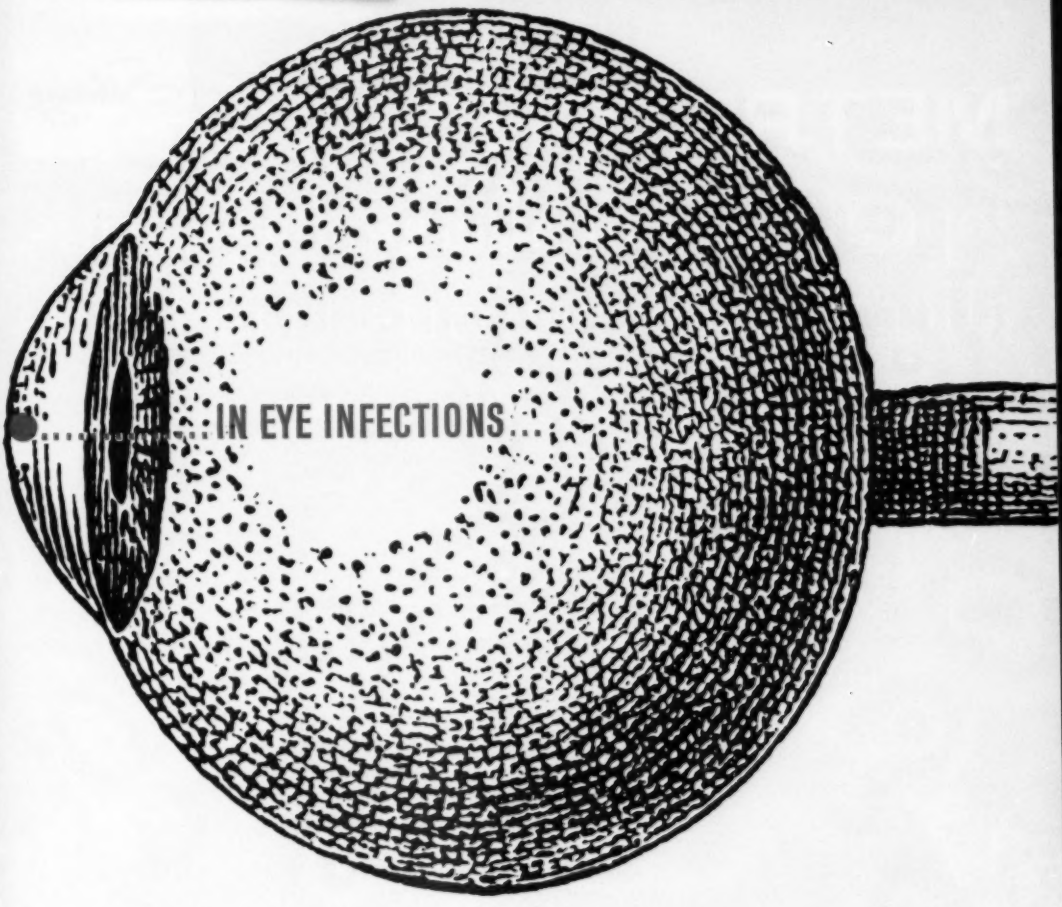

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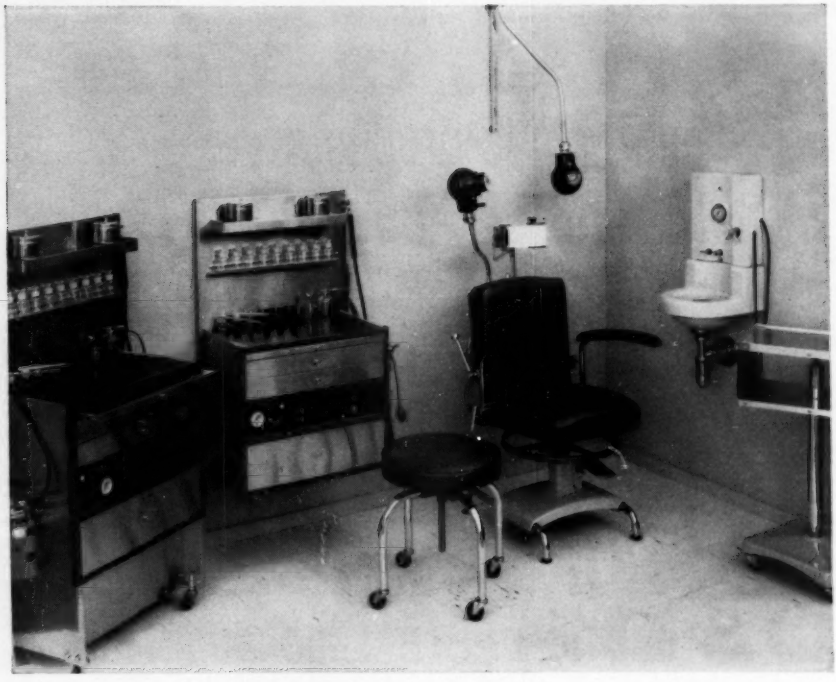

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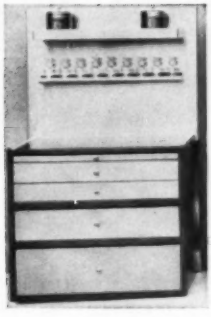

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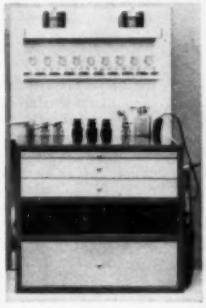

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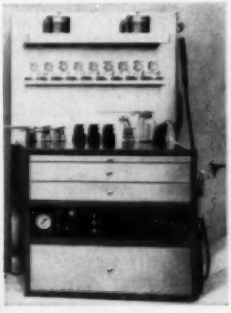

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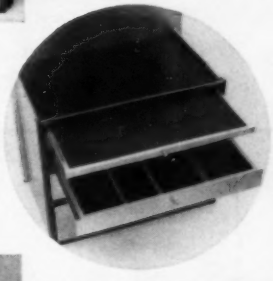

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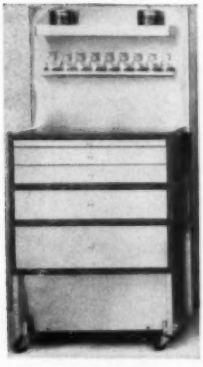

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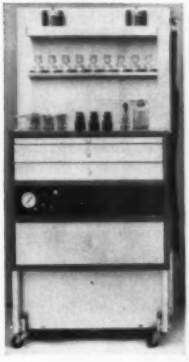

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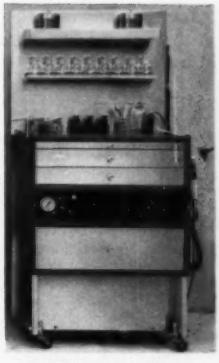
J-6AEF



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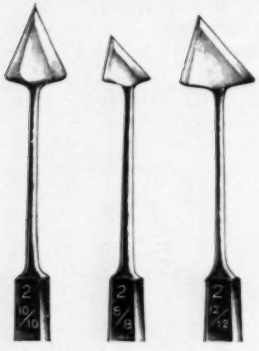

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XVI Jackson Memorial Lecture

SYNDROMES WITH CONGENITAL CATARACT

JULES FRANCOIS, M.D.
GHENT, BELGIUM

IT is indeed an unusual honor to have been invited to give the Edward Jackson Memorial Lecture. Doctor Jackson, who was born in 1856 and died in 1942, was one of the greatest American ophthalmologists. His name was, and is still, held in high esteem, not only in the United States but also across the ocean and all the world over. His book on skiascopy and on refraction, written in 1895, is still a classic and Jackson's cross cylinders are well known everywhere. Since I have read the homage paid to Edward Jackson by those who knew him and his work, and since I have read his papers, which contain so many original ideas, I am stricken with as much admiration as veneration for the physician and the ophthalmologist with his deep wisdom and brilliant erudition; for the investigator and the scientist with his penetrating knowledge and vigorous spirit; for the master and the teacher with his clearness of thought and his analytic mind; for the writer with his great facility of expression, and, above all, for the man with his charming kindness, his attractive modesty, his winning simplicity, his appeasing gentleness and his fundamental goodness.

From the Ophthalmological Clinic of the University of Ghent, Director: Professor Jules Francois.

Presented at the Sixty-Fourth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 11-16, 1959, Chicago.

In many cases the etiology of congenital cataract is obscure. Out of 217 cases of large lens opacities, we have been able to establish the cause in only 70. In 45 cases, there was a hereditary factor; in 12, rubella of the mother; in 6, toxoplasmosis; in 2, galactosemia; in 1, syphilis; in 1, influenza of the mother; and in 1, perhaps sulfonamide poisoning.

To increase the chance of finding a cause, it is, therefore, indispensable not only to consider the maternal and familial antecedents, but also to examine systematically the child affected with cataract. All concomitant anomalies must be revealed if one is to be able to connect them, if possible, with a syndrome to permit the establishment of the genetic nature of the disease. One must bear in mind that a pathologic gene may have a variable expressivity and may also give rise to a pleiotropic, polyphenic or even heterophenic manifestation.

We understand a syndrome as a clinical entity not presenting a series of symptoms involving one single organ or one single system, but characterized by an association of dissimilar anomalies without any apparent interrelationship, since they may involve various organs, various systems and various tissues at one time.

We shall discuss successively Lowe's syndrome, the association of congenital

cataract and oxycephaly, that of congenital cataract and apical malformations, stippled epiphyses, Bonnevie-Ullrich's disease, François' syndrome, congenital hemolytic icterus, Sjögren's syndrome, Marinesco-Sjögren's syndrome, mongolism, and syndermatotic congenital cataracts.

I. LOWE'S SYNDROME

In 1952, Lowe and his co-workers described a syndrome manifesting itself in infants. It was characterized by the following signs and symptoms:

1. *Renal symptoms:* tubular and sometimes glomerular lesions; aminoaciduria, disturbance of ammoniogenesis (very scanty spontaneous ammoniuria and insufficient response to the ammonium salt stress test); albuminuria and intermittent glycosuria

2. *Vitamin-resistant renal rickets,* with pigeon breast, craniotabes, irregular ribs, enlarged epiphyseal extremities, spontaneous fractures, retarded ossification

3. *Retardation in stature and weight* (a certain degree of nanism) and *retarded psychomotor development*

4. *Muscular hypotrophy* with generalized hypotonia and hyporeflexia

5. *Mental deficiency*

6. *Ocular symptoms,* either congenital cataract or glaucoma.

Congenital cataract, which was observed in 10 out of 12 cases, may be either total (Appelmans and co-workers, 1957) or nuclear; it may be combined with cataract of the posterior pole (two cases of Gerard-Lefebvre and co-workers, 1957). When it is total, it is either soft or chalky.

Glaucoma, as observed in 6 out of 12 cases, is either of the congenital type, with buphthalmia (Lowe and co-workers, 1952), or of the simple type (Streiff and co-workers, 1958).

In 4 cases the cataract was associated with glaucoma (3 cases of Lowe and co-workers, 1952; 1 unilateral case of Applemans and co-workers, 1957).

Other ocular symptoms to be noted are searching nystagmus (in 10 out of 12 cases), enophthalmia, divergent strabismus (Streiff and co-workers, 1958), Franceschetti's digital-ocular sign, and atrophy of the iris with photomotor areflexia (Appelmans and co-workers, 1957).

From a chemical point of view, findings were hyperchloremic acidosis, hypophosphatemia with diminution of the alkali reserve; lowering of the phosphorus content and increase of the alkaline phosphatase content.

Other manifestations observed were intermittent fever, trembling crises, stridor, digestive disorders with vomiting, electroencephalographic changes, skin anomalies (eczema, dry and rough skin), general malformations (camptodactylia, pes cavus, cryptorchism, sunken or flat chest, hernias, cox plana, macrognathia, anomalies of the external ear, arched palate, microdontia, retrognathia, saddle nose, hypertelorism, brachycephaly with precocious closure of the sutures, parietal, occipital and frontal bulgings, internal hydrocephaly).

Lowe's disease, of which 12 cases are now known, seems to be due to a hereditary factor. Gerard-Lefebvre and his co-workers (1957), as a matter of fact, observed the familial occurrence of the syndrome. Two brothers were affected, and also a cousin; the mothers were sisters. The brother who died from Schlesinger's disease (1956) probably showed the same affection. The parents of the patient of Debre and his co-workers

(1955) both had marked amino-aciduria: 13 amino-acids for the father, 12 for the mother. The father of the patient of Streiff and his colleagues also had amino-aciduria (9 amino-acids).

Considering that all the patients known up to now (12) are males, Streiff (1958) reached the conclusion that there was sex-linked recessive heredity. Franceschetti (1958), however, believes that in view of the small number of cases, autosomal heredity limited by sex cannot be excluded.

FIG. 1—Congenital cataract and oxycephaly.

II. CONGENITAL CATARACT AND OXYCEPHALY

The association of congenital cataract with oxycephaly (fig. 1), less frequently with plagiocephaly, constitutes a real syndrome, which has repeatedly been mentioned: Groenouw (1901), Kraus (1902), Alexander (1903), Patry (1905, 3 cases), Aubaret and Guillot (1934), Lartigue (1939, 2 cases), Sourdille and co-workers (1939, 1946, 2 cases), Gänsslen (1940), Falls (1943, 2 cases), Walsh (1947), Renard and Laporte (1951), Vest (1955). There is usually a zonular cataract, which may be accompanied by other ocular manifestations, due to oxycephaly (optic atrophy, exophthalmia, etc.).

III. CONGENITAL CATARACT AND APICAL MALFORMATIONS

The combination of congenital cataract and apical anomalies, which is often familial, has been described by numerous authors: Appenzeller (1884, polydactyly), Ginestous and Lavie (1903, syndactyly), Manson and Nettleship (1912, polydactyly), Koby (1923, camptodactyly), Rieger (1941, polydactyly). In a case described by Karsch (1937), the congenital cataract was associated with tapetoretinal degeneration and apical dystrophy of the "lobster-claw" type. The tapetoretinal degenerations may be accompanied by congenital cataract, especially in Bardet-Biedl's syndrome, of which polydactyly is the classical symptom.

IV. CONGENITAL STIPPLED EPIPHYSES

Stippled epiphyses, also called Contradi's syndrome (1914), congenital calcareous or calcifying chondrodystrophy (Hünermann, 1931), dysplasia epiphysialis punctata or chondrodystrophic epiphyseal fetal calcinosis, is a congenital affection which manifests itself from early infancy, in the first few days or months after birth.

The very high mortality in the first two years of life and the regressive development of the disease if the patient survives, explain why the diagnosis is rarely made after the age of two. The affection is rare; only about 50 cases of it are known.

Symptomatology

The clinical and radiologic signs, which are of primary importance, are accompanied by various dystrophies, and also by a bilateral total congenital cataract.

Clinical signs

1. The shortening of the long bones is characteristic. Whereas the length of the trunk is normal, there is a more or less marked micromelia, which reduces the height considerably (figs. 2 and 3).

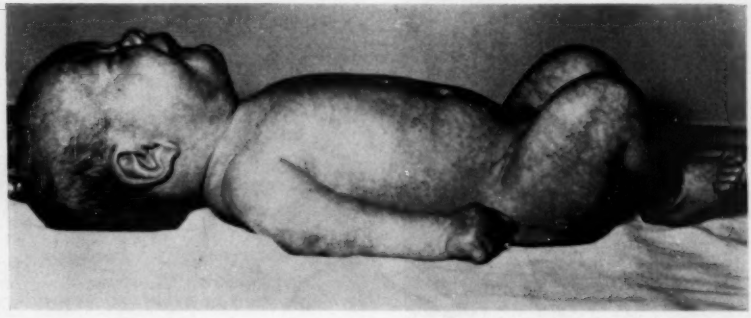

FIG. 2—Stippled epiphyses with congenital cataract.

This micromelia is not harmonious; it shows definite rhizomelic predominance and gives the newborn the appearance of achondroplasia (disproportionate nanism). It involves the legs more than the arms, and the proximal parts more than the distal. Moreover, it is often asymmetrical, i.e., the reduction in length of the limbs on one side is more pronounced than on the other (Jeune et al., 1953).

2. Morphologic anomalies of the bones are often observed: enlargement of the metaphyseal extremities; incurvation of some diaphyses, especially of the femur; kyphoscoliosis; digital anomalies; malformations of the feet, etc. (Resnick, 1943; Duffy and Vinke, 1947; Ford, Schneider and Brandon, 1951; Schönenberg and Shallock, 1953).

3. In half of the cases there is articular dysfunction (fig. 4), with abnormal posture in flexion, limitation or abolition of movements, and pain on passive movement, involving one or several large joints of the limbs (knee, hip, elbow), sometimes the joints of the hands and fingers, rarely those of the neck. These articular anomalies are not always due to bone malformations, but are sometimes caused by a fibrous change in the muscles (Lund, 1943).

Radiological signs

1. The roentgenograms of the skeleton show *epiphyseal calcifications*, which constitute the main sign of the affection. These calcifications are more or less regularly distributed between the metaphyses and the articular surfaces. They are often very numerous and fairly dense, either round or angular in shape, and their size ranges from that of a pin's head to bird shot, rarely reaching the size of a cherry stone. They may remain localized in a portion of a limb or in some joints (Swoboda, 1950), but

FIG. 3—Stippled epiphyses and congenital cataract.

generally, and even in the absence of clinical signs, they are dispersed throughout the skeleton, especially in the epiphyses of the long bones, with a marked predilection for the extremities of the humerus and femur (figs. 5, 6, 7, 8, and 9). Apart from these calcified spots, the epiphyses are absent or enlarged and deformed.

2. Another frequent roentgenographic finding is that of *metaphyseal deformities,* in the shape of a trumpet, a spur or a mushroom (Bateman, 1936), combined with anomalies of the ossification line, which is irregular, convex, concave or markedly oblique.

These cardinal signs are often accompanied by *other dystrophic manifestations.*

1. *Cutaneous manifestations:* Thickening of the skin with hypertrophy of the panniculus adiposus, rough and scaly skin (Lightwood, 1931; Kwerch, 1950), ichthyosis (Contradi, 1914; Hässler and Schallock, 1953; Jeune et al., 1953; Armaly, 1957; obs. pers., 1959), desquamating dermatitis (Burckhardt,

FIG. 4—Stippled epiphyses and congenital cataract.

FIG. 5—Stippled epiphyses. Upper limb (shoulder and elbow).

1938), adherence of the palmar skin to the deep layers (Bateman, 1936), hair anomalies: few and rudimentary hairs, eyebrows and eyelashes (Lund, 1942; Jeune et al., 1953; obs. pers. 1959).

2. *Craniofacial anomalies:* microcephaly (Hässler and Schallock, 1949; Schönenberg and Schallock, 1953), megacephaly (obs. pers., 1959), saddle nose (de Toni and Papio, 1948; obs. pers., 1959), aplasia of the mandible (obs. pers., 1959), cleft palate.

3. *Congenital cardiopathies:* Interauricular and interventricular communication (Schönenberg and Schallock, 1953), truncus arteriosus (Tisdall and Erb, 1924), persistent ductus arteriosus

(Lightwood, 1931); Roger's disease (Borovsky and Arendt, 1944).

4. *Other malformations* have also been noticed: clubfoot, acromicria, syndactyly, camptodactyly, kyphosis (obs. pers., 1959), wedge vertebrae, renal malformations, wolf's maw (gueule de loup), etc.

5. *General debility:* gastrointestinal disturbances, mental retardation.

the age of 11 months; in de Toni and Papio's case [1948], at 4.5 months). In a single case (Jolly, 1951), it was unilateral.

It has been described by Contradi (1914), Lightwood (1931), Bateman (1936), Bloxsom and Johnston (1938), Maitland (1930), Lund (1942), de Toni and Papio (1948), Ford et al. (1951), Jolly (1951), Scott (1952), Jeune et al. (1953), and Armaly (1957).

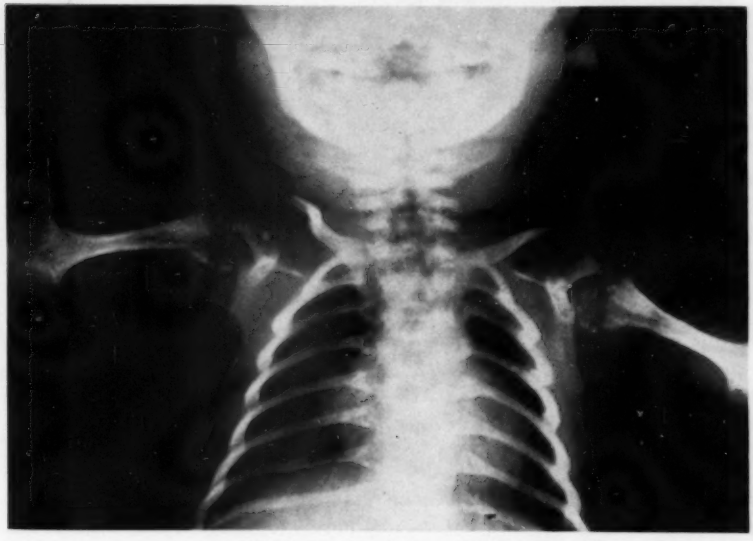

FIG. 6—Stippled epiphyses. Shoulder.

6. The *congenital cataract* is, after all, the sign which interests us most of all. It has been observed in 50 per cent of the cases and consequently has great diagnostic value. It has never been found in ordinary achondroplasia.

The cataract is bilateral, usually total, sometimes nuclear (obs. pers., 1959), with radial opacities. It is usually congenital but may, in exceptional cases, develop during the first year of life (in Bloxsom and Johnston's case [1938], at

On the other hand, it is probable that the cataract has not been perceived in a number of cases because an ophthalmologic examination has not always been carried out.

Hervouet has been kind enough to lend us microphotographs of a cataract in a child with stippled epiphyses (figs. 10 and 11). They show diffuse degeneration of the lens fibers; at the anterior pole is a homogenized area; globules ex-

FIG. 7—Stippled epiphyses. Left knee.

tricated from the fibers are between the lens and the capsule.

Congenital cataract is exclusively observed in the complete form with definite chondrodystrophy and rhizomelic micromelia. This fact lends it a prognostic value, because in nearly all the cases where it was found, except those of Jeune and his colleagues (1953) and Armaly (1957), death ensued before the age of 2 years.

In the cases with cataract, skin changes are the most frequent.

In the incomplete or minor, almost purely radiologic form, cataract has never been established.

In three cases, discission of the cataract was performed. In the case of Bloxsom and Johnston (1938), a dense and opaque membrane had formed; in those of Jeune and his colleagues (1953) and of Armaly (1957), the result was satisfactory.

Other ocular anomalies observed were bilateral optic atrophy in two cases (Jolly, 1951); partial, unilateral cataract;

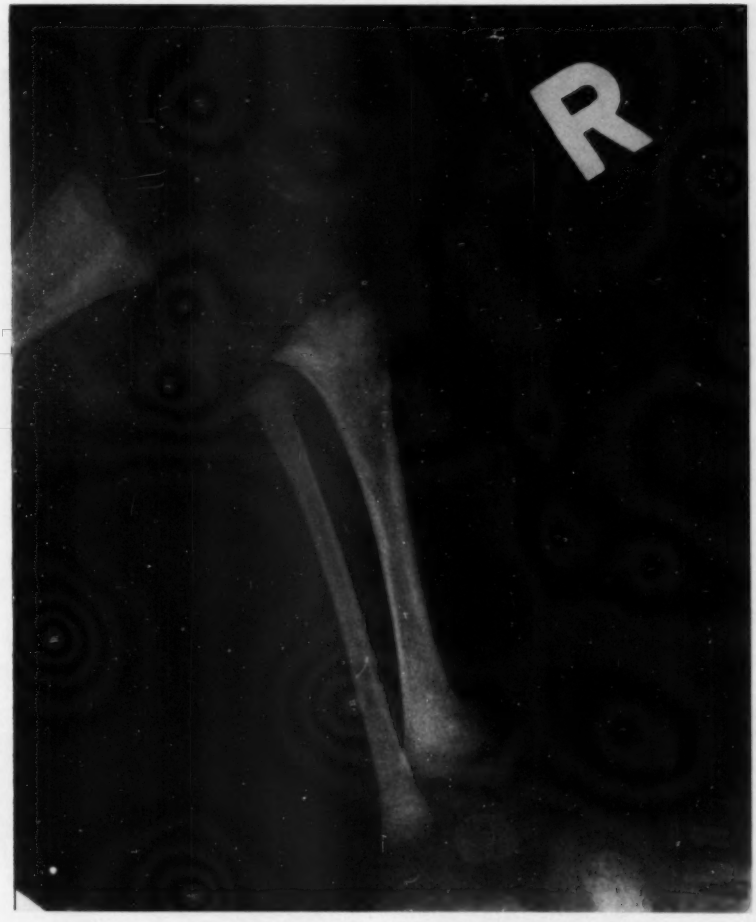

FIG. 8—Stippled epiphyses. Right knee and foot.

cataract absorbed after discission (Armaly, 1957). In this last-mentioned case, the retina had a granular aspect.

Heredity

The frequency of fetal epiphyseal calcinosis is estimated at 1 in 500,000 (Schönenberg and Schallock, 1953),

Girls are more often affected than boys (4:3); the female predominance is even more marked in the cases with congenital cataract (4:1).

A concordant manifestation in the two partners of a pair of identical twins was observed by Resnick (1943). This fact speaks definitely in favor of a genetic factor, especially since three instances of familial cases in siblings are known: complete form in two sisters (Maitland, 1939); minor form in four brothers and

FIG. 9—Stippled epiphyses. Right knee.

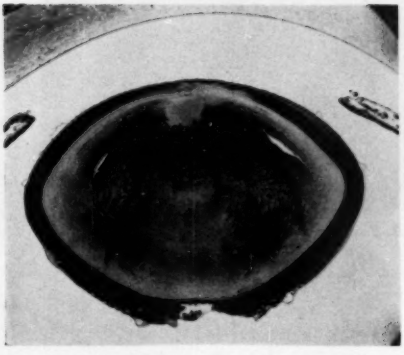

FIG. 10—Congenital cataract with stippled epiphyses. Diffuse degeneration of lens fibers (Hervouet)

FIG. 11—Congenital cataract with stippled epiphyses. Anterior pole. (Hervouet)

sisters (Raap, 1943); minor form in a brother and a sister (Vinke and Duffy, 1947). Among siblings the clinical form is always the same.

If, on the other hand, it is considered that consanguinity of the parents is relatively frequent (Tisdall and Erb, 1924; Lightwood, 1931; Bateman, 1936; de Toni and Papio, 1948; 4 out of 12 cases according to Fraser and Scriver, 1954, i.e., 33 per cent), simple recessive heredity has to be assumed.

It should, however, be borne in mind that Bateman (1936) found brachymelia of the humerus in the mother and the

grandfather; Hünermann (1931), brachymelia of the femur in the father. Resnick (1943) observed an incomplete form of the disease in the mother. Maitland (1939) and Jorup (1944) found digital anomalies resembling those of the dysplastic children in one of the ancestors. These cases, to which we may add a personal case, demonstrate the possibility of direct transmission, in particular since the regressive nature of the epiphyseal calcifications and of the brachymelia makes it unlikely that clinical or radiological signs will be found in the ancestors.

V. THE BONNEVIE-ULLRICH SYNDROME

The syndrome of Bonnevie (1935) and Ullrich (1930) is comprised of a great number of congenital symptoms, of which the whole or a part may be discovered in varying combinations. These symptoms may be unilateral (asymmetrical form) or bilateral (symmetrical form):

1. The principal symptom is pterygium colli, a broad cutaneous fold which extends from the mastoid to the acromion and is accompanied by horizontal cutaneous pads on the nape of the neck, which is short and thick. One may also find pterygiums at the joints, especially at the elbows and knees, causing anomalies in the position of the limbs and limitation of their movements.

2. There is a laxity and hyperelasticity of the skin. The skin is not adherent to the subjacent planes in certain places, most often the neck and the buttocks (cutis laxa).

3. There exists a generalized muscular hypotonia with atrophy of certain muscles, most often of the pectorals, as well as a hyperlaxity of the ligaments.

4. One also notices a hypertrichosis, especially of the face and thorax, as well as a low implantation of the hairs.

5. A facial paralysis and ocular paralyses (especially of the sixth nerve) are not rare.

6. During the first years there is a lymphangiectatic edema, especially on the backs of the extremities and in the neck.

7. Disturbance of skeletal development with dyscrania and nanism is frequent.

a. There exists a dyscrania with enlargement of the fontanelles and separation of the sutures. The base of the cranium is strongly sloping, which gives rise to a low position of the auricle and the external auditory canal. The digitiform impressions are often pronounced.

b. The face also presents anomalies: hypertelorism, epicanthus, ptosis, flattening of the root of the nose, cup-shaped depression of the point of the nose, hypoplasia of the mandible, arched palate, harelip or wolf's maw (gueule de loup). The dystrophic aspect of the face, together with the pterygium colli, gives the patient a "sphinx's head."

c. One may note anomalies of the foot (clubfoot), or of the hand (syndactyly, clinodactyly, camptodactyly, arachnodactyly, interdigital webbing). The nails are dystrophic, brittle, spoon-shaped (koilonychia). It is not rare to see a cubitus valgus, a subluxation of the radial head, or a dislocation of the hip.

d. The thorax is funnel-shaped and the nipples are far apart.

e. Radiographic examination shows a broadening of the epiphyses with mushroom deformity.

8. One often notices a slight mental backwardness with psychomotor retardation.

9. Other anomalies reported: umbilical hernia, situs inversus, congenital cardiopathies, duodenal stenosis, malformations of the external genital organs (hypospadias in the boys, overdevelopment of the clitoris in the girls).

10. In women affected by the symmetrical forms, one may also observe an ovarian agenesia with hypersecretion of the gonadstimulins (Turner's syndrome, 1938).

The infantilism which results may be dissociated; the sexual characteristics dependent on the estrogens (breasts, labia minora, vagina, uterus) remain infantile, while the sexual characteristics which depend on the androgens (labia majora, hairs of the axilla and pubis) develop. There is a primary amenorrhea. Chromosomal determination of the sex gives a "masculine" result in patients whose sex is apparently feminine.

11. In addition to the ocular anomalies already pointed out, one may note an absence of the caruncle, a hypoplasia of the lacrimal glands, an exophthalmia, pigmentary anomalies of the retina and, especially, a congenital cataract (Beyer et al., 1956).

The pathogenesis of the affection has not yet been established, but, although an environmental cause cannot be excluded, certain observations favor an hereditary factor:

1. Predominance in members of the feminine sex is obvious: there are four women affected to one man (Ullrich, 1950).

2. Liebenam (1938) observed a Bonnevie-Ullrich status, unilateral and mirror-image, in a pair of monozygotic twins. Ostertag and Spaich (1936) noticed a typical syndrome in one of a pair of univitelline twins, with digitiform impressions and a web between the second and third toes in the other.

3. With regard to family observations, Teveli (1930) saw a web between the third and fourth toes of the father of an affected child. Kieser (1939) noticed a pterygium colli in ten members of one family (three generations). Baumann (1945) observed three first cousins with a congenital angiectatic lymphedema. Rossi (1945) published the report of a case of Bonnevie-Ullrich's syndrome in which a hypertelorism existed in the maternal line, and a polydactyly in the paternal line. Rossi and Howald (1947) described two sibships: several of the members of one of these families were affected by a status dysraphicus rather than by a status Bonnevie-Ullrich, while in the other a woman and her paternal female cousin presented a typical Ullrich syndrome. Cotterman (1948) reports strabismus and an epicanthus in the brother, mother and maternal male cousin of an affected subject. Foucar (1948) observed in two sisters a syndrome of Klippel-Feil, rather than of Bonnevie-Ullrich.

Wertheman and Reiniger (1949) report the case of a mother who gave birth to two stillborn fetuses with the affection and a third child, born at term, with a congenital cardiopathy. Cotterman and Falls (1950) observed the syndrome in two sisters.

It is difficult to draw conclusions of a definitive nature from these observations, but it certainly seems that the gene in question presents an irregular dominance with incomplete penetrance and variable expressivity.

VI. DYSCEPHALY WITH BIRD-FACE AND DENTAL ANOMALIES, NANISM HYPOTRICHOSIS, CUTANEOUS ATROPHY, MICROPHTHALMIA AND CONGENITAL CATARACT

Based on twenty-four reports in the literature, described under various names, and on two personal observations, to which we are now able to add two others (Larmande, 1959), we have described a syndrome (1957, 1958, 1959) which is characterized by seven essential symptoms.

1. Dyscephaly, which comprises, on the one hand, a dyscrania and, on the other, a mandibulofacial dysmorphia:

a. The dyscrania consists sometimes of a scaphocephaly, with the cranium shaped like the keel of a ship with protruding forehead. Often we see a brachycephaly with platybasia. Scaphocephaly was observed by Aubry (1893); the brachycephaly with platybasia by Hallermann (1948), Marchesani (1949), Ullrich and Fremerey-Dohna (1953), Nizetic (1954), Leffertstra (1956), Larmande (1959), and by us (2 cases, 1959, fig. 12). An occipital bulging was noticed by Bergmeister (1911) and by Ludwig and Korting (1950); a frontal bulge (prominence of the frontal protuberances) by Schondel (1943, case 1), Marchesani (1949), Ullrich and Fremerey-Dohna (1953), Leffertstra (1956), and Gregersen (1956); a vaulting of the parietal bones by Bergmeister (1911), Schondel (1943, case 2), Ludwig and Korting (1950), Ullrich and Fremerey-Dohna (1953), Weyers (1954), to such a degree that the cranium took the shape of a square (Schondel, 1943, case 1), or of a trapezium (Ullrich and Fremerey-Dohna, 1953; Weyers, 1954). The forehead is sometimes narrow, elevated and receding (Streiff, 1950; Ludwig and Korting, 1950; Leffertstra, 1957; personal case 2); the malar bone may be aplasic (Ludwig and Korting, 1950; Weyers, 1954).

While there is sometimes microcephaly (Moehlig, 1946; Streiff 1950, personal observations) there may also be relative hydrocephaly (Schondel, 1943, case 1).

There are often dehiscences of the lambdoid and longitudinal sutures (Bergmeister, 1911; Moehlig, 1946; Ludwig and Korting, 1950; Ullrich and Fremerey-Dohna, 1953; Nizetic, 1954) and

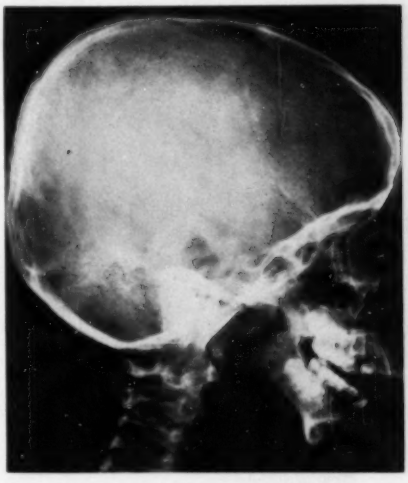

FIG. 12—Brachycephaly with platybasia.

open fontanelles (Bergmeister, 1911; Ullrich and Fremerey-Dohna, 1953; Gregory, 1955, case 1; personal observation 2).

Nizetic (1954) reports a thinning of the cranial box and a partial calcification of the roof of the cerebellum, accentuated digitiform impressions, a perisellar hyperostosis and an interclinoid bridge.

In our patient there was a hyperostosis around the cranial sutures and a broad synchondrosis between the sphenoid and the occipital bone.

b. The mandibulofacial malformation is characterized above all by mandibular aplasia, which is a striking feature in every case (fig. 13). Sometimes the maxilla also has a slight degree of hypoplasia (personal observations).

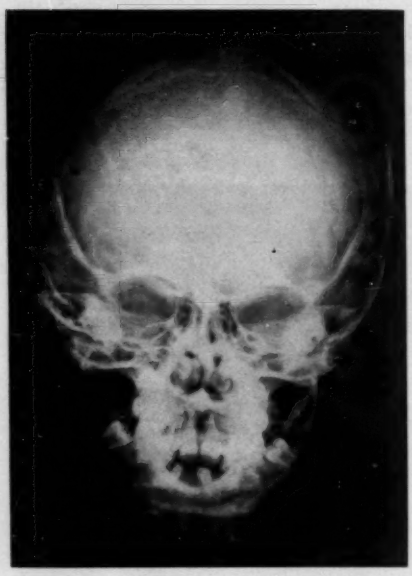

FIG. 13—Mandibular aplasia with hypoplasia of the maxilla.

The nose is thin, tapering and pointed (Bergmeister, 1911; Schondel, 1943, cases 1 and 2; Moehlig, 1946; Hallermann, 1948, cases 1 and 2; Streiff, 1950; Ludwig and Korting, 1950; Ullrich and Fremerey-Dohna, 1953; Weyers, 1954; Leffertstra, 1956; Larmande, 1959; personal observations 1 and 2), accompanied by hypoplasia of the nasal bone (Weyers, 1954), which is often curved and shaped like a parrot beak (Bergmeister, 1911; Schondel, 1943, case 1; Moehlig, 1946; Hallermann, 1948; Streiff, 1950; Leffertstra, 1956; Gregersen, 1956; Blodi, 1957, cases 1 and 2; Larmande, 1959; personal observation 1).

The nasofrontal angle is more acute than normally (personal observation 1). The palate is generally vaulted (Schondel, 1943, case 2; Moehlig, 1946; Streiff, 1950; Ludwig and Korting, 1950; Ullrich and Fremerey-Dohna, 1953; Nizetic, 1954; Leffertstra, 1956; Larmande 1959, personal observation). Nizetic (1954) and Blodi (1957, case 2) have mentioned a facial asymmetry from hemiatrophy.

The dyscephaly with aplasia of the mandible and thin curved nose gives the patient a birdlike profile, especially that of a parrot (fig. 14, 15 and 16). The frequent existence of a double cutaneous chin with a central cleft contributes to giving the patient a characteristic physiognomy (Hallermann, 1948, case 2; Streiff, 1950; Gregersen, 1956, case 2; Blodi, 1957, case 2; Larmande, 1959; personal observations 1 and 2).

2. The dental anomalies are, so to speak, constant: some of the teeth may be lacking (Bergmeister, 1911; Blodi, 1957; Larmande, 1959; personal observation, fig. 17); those that are present are often malformed (Moehlig, 1946; Hallermann, 1948, case 1 and 2; Nizetic, 1954; Gregory, 1955; Larmande, 1959) or irregularly implanted (Aubry, 1893; Schondel, 1943, cases 1 and 2; Moehlig, 1946; Marchesani, 1949; Streiff, 1950; Ludwig and Korting, 1950; Blodi, 1957; Larmande, 1959; personal observation). It is not rare to observe an open bite (Ludwig and Korting, 1950; Larmande, 1959; personal observation, fig. 18) or a hiatodontia (Weyers, 1954). Caries are frequent and early (Schondel, 1943, cases 1 and 2; Moehlig, 1946; Nizetic, 1954; Leffertstra, 1956).

FIGURE 14.

FIGURE 15.

FIG. 16—Case of Larmande.

FIGURE 17.

FIGURE 18.

FIG. 19—Case of Larmande.

3. Harmonious and well proportioned nanism is reported in 20 cases out of 27, body height being 11 to 24 cm. below the normal (fig. 19). In six cases the height is not given (Aubry, 1893; Hallermann, 1948, case 2; Marchesani, 1949, cases 1 and 2; Ludwig and Korting, 1950; Ullrich and Fremerey-Dohna, 1953). Only one report (Weyers, 1954) specifically mentions absence of nanism. This nanism is accompanied with a retardation in skeletal development (personal observation).

4. Hypotrichosis is absent only in Nizetic's case (1954). It exists in all the other cases. It involves the axillary and pubic hair (Leffertstra, 1956; personal observation) as well as the hairy skull, eyelashes and eyebrows (figs. 20, 21 and 22).

FIGURE 20.

The hair is fine, thin, clear, rarely whitish (Schondel, 1943; Gregory, 1955) and generally absent in places

FIG. 21—Case of Larmande.

FIG. 22—Case of Larmande.

(alopecia areata). The bald patches are often localized on the forehead (Marchesani, 1949; Blodi, 1957, case 2), on the posterior and temporal aspect of the skull (personal observation 1), or along the cranial sutures (sutural alopecia: Aubry, 1893; Bergmeister, 1911; Ullrich and Fremerey-Dohna, 1953; Weyers, 1954).

The hairs in the eyebrows are thin and scattered, and sometimes completely lacking (Schondel, 1943, case 1; Gregory, 1955; Blodi, 1957, cases 1 and 2; personal observation 2). The eyelashes are short, fine, few in number, and sometimes absent (Schondel, 1943, case 1; Moehlig, 1946; Weyers, 1954).

5. Cutaneous atrophy usually involves the head only. The skin is thin, white, dry, soft, taut, sclerous and veined. (Aubry, 1893; Schondel, 1943, cases 1 and 2; Moehlig, 1946; Weyers, 1954; Gregory, 1955; Gregersen, 1956; Blodi, 1957; Larmande, 1959; personal observation 2). Cutaneous atrophy is particularly marked on the nose and is often confined to this area (Schondel, 1943, case 2; Hallermann, 1948, cases 1 and 2; Streiff, 1950; Ludwig and Korting, 1950; Ullrich and Fremerey-Dohna, 1953; Nizetic, 1954; Larmande, 1959; personal observation 1).

Hallermann (1948, case 2) noted numerous cutaneous folds in the face; Ludwig and Korting (1950), a vitiligo on the trunk; and we, a livedo on the thighs and the popliteal spaces.

6. Bilateral microphthalmia was noted in 22 cases out of 27. It is not mentioned by Aubry (1893), Moehlig (1946), Nizetic (1954) or Gregory (1955, cases 1 and 2). It is of variable degree, the corneal diameter varying from 7 mm. (Schondel, 1943, case 2; Gregersen, 1956, case 2) to 10 mm.

7. A bilateral congenital cataract, complete or incomplete, is reported in all the cases, except that of Schondel (1943, case 1), in which there were sequelae of uveitis.

In Gregersen's patient (1956, case 2), no lens opacity was noted at the age of 5 years, but at the age of 15 there was a spontaneous absorption of the lens with membraneous conversion of both lenses.

The congenital cataract is generally constituted by liquefied lens masses, discission of which gives rise to the outflow of a milky-white fluid (Bergmeister, 1911; Ullrich and Fremerey-Dohna, 1953; Leffertstra, 1956; Gregersen, 1956; personal observation 1).

Spontaneous absorption of the lens on the other hand is a relatively frequent phenomenon (Hallermann, 1948; Gregersen, 1956, case 2; Blodi, 1957, case 1; personal observation 2).

In addition to microphthalmia and congenital cataract, other ocular anomalies have been reported that are less important or rarer; blue sclera (Schondel, 1943, case 2; Moehlig, 1946; Ludwig and Korting, 1950; Weyers, 1954; Nizetic, 1954); distichiasis (Blodi, 1957, case 1); ptosis (Ludwig and Korting, 1950); antimongoloid slanting of the palpebral fissures (Ludwig and Korting, 1950; Ullrich and Fremerey-Dohna, 1953; Blodi, 1957, case 1); notching of the edge of the outer third of the lower eyelid (Ludwig and Korting, 1950); corneal vaulting (Gregersen, 1956, cases 1 and 2), atrophy of the iris (Ludwig and Korting, 1950); posterior synechias (Ullrich and Fremerey-Dohna, 1953), floating bodies of the vitreous (Weyers, 1954), ocular hypertension (Gregersen, 1956, case 2); changes in the ocular fundus; small disseminated pigmentations (Gregersen, 1956, case 1), foci of chorioretinal atrophy (Streiff, 1950; personal observation 1), peripapillary choroid atrophy (Hallermann, 1948; cases 1 and 2), cherry red macula surrounded by a greyish zone (Nizetic, 1954), pale discs (Gregersen, 1956, case 1), coloboma at the entrance of the optic nerve (personal observation 1), macular retinopathy (personal observation 2) (fig. 23). Given the feeble vision of subjects affected, it is not surprising that the majority of them have a horizontal nys-

FIGURE 23.

tagmus, rotatory or pendular, as well as a convergent or divergent strabismus.

Some general symptoms have been reported in some instances but these are exeptional: underdevelopment of the subcutaneous adipose tissues and of the musculature (Schondel, 1943, case 1); osteoporosis (Moehlig, 1946; Nizetic, 1954); hypogenitalism with or without cryptorchism (Ludwig and Korting, 1950; Nizetic, 1954); clawhand (main en griffe), (Nizetic, 1954); syndactyly (Gregory, 1955, case 2); coxalgia (Gregory, 1955, case 2); platyspondylia (Nizetic, 1954); slight mental backwardness (Moehlig, 1946; Nizetic, 1954), dysraphic signs; lordosis with or without scoliosis (Schondel, 1943, cases 1 and 2; Ludwig and Korting, 1950; Nizetic, 1954); spina bifida (Schondel, 1943, case 2; Nizetic 1954); funnel thorax (Nizetic 1954), and hypotrophy of the muscles of the scapular girdle (Nizetic 1954).

Heredity

Apparently heredity is not a factor. All the cases described in the literature are unique. No consanguinity of the parents has been reported. However, these facts do not necessarily exclude a hereditary factor. In point of fact, it may be a question either of a recessive affection or of a mutation.

As the patients have no descendants so far as is known at present, it is impossible to come to any conclusion. Nevertheless, the identical morphological features of all the subjects affected is strongly in favor of a hereditary factor. This is all the more likely as concordance in the two members of a couple of identical twins has just been observed (Waardenburg, 1958). Larmande (1959) has also observed several cases in one family.

VII. CONGENITAL HEMOLYTIC ICTERUS

This affection is characterized by an icterus with splenomegalic anemia. The icterus is due to a massive destruction of the erythrocytes during hemolygic crises. Hematologic examination shows a microspherocytosis, a reticulocytosis, and a diminution of the osmotic resistance of the red cells. There are skeletal alterations: osteoporosis, thinning of the cortex of the bones, and protrusion of the frontal and parietal bones. Gänsslen (1940) has drawn attention also to an oxycephaly, apparently existing in 50 to 60 per cent of cases. There exist *formes frustes* or latent cases characterized by an abnormal fragility of red cells (Gänsslen, 1940; Race, 1942). Splenectomy is a very effective treatment.

Ocular manifestations are not exceptional. Narrow or mongoloid palpebral fissures, microphthalmia, heterochromia of the iris, myopia, a dyschromatopsia and congenital cataracts have been reported.

The heredity of congenital hemolytic icterus is shown by its concordant manifestation in identical twins (Debre et al., 1937; Culbreath, 1937; Debre et al., 1945).

The affection is dominant with irregular penetrance (Campbell and Warner, 1926; Race, 1942; Gasser, 1951; Young et al., 1951; Bernard et al., 1952).

VIII. SJÖGREN'S SYNDROME

In 1935 Sjögren drew attention to the frequent association of congenital cataract and oligophrenia—one might even speak of a veritable correlation between them.

1. The congenital cataract is always bilateral, generally zonular, sometimes polar or total. Franceschetti and Brugger out of 18 cases found a zonular cataract eleven times, a total cataract four times, an anterior polar cataract twice, and once a posterior cortical cataract. When the cataract is partial, it often happens that it develops and becomes total (Dufour et al., 1954). It is not always strictly congenital, but exceptionally may develop in the course of the first months or years of life (Dufour et al., 1954).

2. Oligophrenia comprises not only mental weakness, but also imbecility and idiocy.

Sjögren found the combination of the two affections in 44 cases in 30 families, and has shown that it is a true clinical entity. Waardenburg (1919) and others had already published similar cases, but after Sjögren's work the number of observations multiplied (von Hofe 1936, 5 cases; Heuyer et al., 1941; Rochat, 1941; Falls, 1943; Franceschetti and Brugger, 1944, 36 cases; Krause, 1946; Heuyer et al., 1947; Guillaumat et al., 1948; Bagley, 1949; Spitzer and Mann, 1950; Pfändler, 1950; Schachter, 1950; Cordes, 1951; Euziere et al., 1951, 2 cases; Gursdorf et al., 1952; Christiane Vignier, 1952; Dufour et al., 1954, 14 cases; personal observation).

Madame Schappert-Kimmijser (1958) found an oligophrenia in 18 children out of 75 blinded by congenital cataract (24 per cent) and in 7 out of 88 children who were amblyopic because of congenital cataract (8 per cent).

Mental retardation often does not manifest itself until the child is four years old or more (Krause, 1946). It was sometimes believed that this retardation was due to the absence of vision. Although it is certain that some patients improve both from the mental and intellectual point of view after a successful operation, it nevertheless remains true that the greater part of the physical deficiency persists (Falls, 1943). Moreover, we all know of congenitally blind persons who develop quite normally from the mental point of view.

Sjögren's syndrome may be associated with other ocular and general anomalies:

1. Similar ocular anomalies: nystagmus, microphthalmia (in about 12 per cent of cases), more rarely with megalocornea, myopic choroiditis, detachment of the retina, optic atrophy, keratoconia, ectopia of the lens (Franceschetti and Brugger, 1944; Pfändler, 1950).

2. Other anomalies of a general nature: Scheuermann's disease (juvenile vertebral epiphysitis, epilepsy, arachnodactylia or brachymorphia, skeletal malformations, infantile hemiplegia, deafness, cryptorchidia, spina bifida, gueule de loup (Dufour et al., 1954).

Sjögren's syndrome is transmitted by autosomal recessive inheritance, by the intermediation of a single polyphene gene of variable expressivity, which produces a true encephalo-ophthalmic dysplasia:

1. There are no other cases in direct descent or ascent.

2. Affection of collaterals (first cousins above all) (Dufour et al., 1954).

3. The affection of several members of the same family is not rare (Sjögren, 1935; Franceschetti and Brugger, 1944; Dufour et al., 1954). The risk for first cousins of affected subjects appears to be about 16 per cent (Sjögren, 1935).

4. Consanguinity of the parents is frequent: the percentage is probably about 6.7, according to Franceschetti and Brugger (1944) and 14 per cent according to Dufour and his colleagues (1954), whereas the percentage for the general population does not exceed 1.5.

5. In view of the fact that women are affected as well as men (18:26, according to Sjögren, 1935), a heredity bound to sex can be excluded.

IX. THE SYNDROME OF MARINESCO-SJÖGREN

Another syndrome, much rarer than that of congenital cataract and oligophrenia, is characterized by the *combination of congenital cataract, spinocerebellar ataxia and mental retardation.*

Marinesco and his colleagues (1931) were the first to draw attention to this new clinical entity, which they observed in four first cousins of a sibship of six. In 1947 and 1950 Sjögren made a thorough clinical and genetic study of this syndrome. He was able to collect 14 cases, distributed throughout 6 families. Since then, other observations have been published: Garland and Moorhouse, 1953; Dufour and his co-workers, 1953, 1 case; Franceschetti and his colleagues 1956, 1 case.

The spinocerebellar ataxia may be replaced by a spastic diplegia (Sjögren, 1950) or by the peroneal muscular atrophy of Charcot-Marie-Tooth (Dufour et al., 1954), affections within the frame of ataxias from spinopontocerebellar degeneration.

Richards (1950) has published the report of a case similar in some respects but, nevertheless, very different from the neurological point of view.

The syndrome of Marinesco-Sjögren may be associated with other congenital anomalies, more particularly anomalies of the skeleton: short stature, scoliosis, genu valgum, limitation of extension movement of the knee joints, anomalies of the fingers and toes, retardation of somatic development.

The syndrome is hereditary and is transmitted by the autosomal recessive mode. Consanguinity of the parents is also frequent (Sjögren, 1950; Garland and Moorhouse, 1953; Franceschetti and colleagues, 1956). Marinesco and his colleagues observed the syndrome in four first cousins; Garland and Moorhouse (1953), in two cousins who were the offspring of marriage of two sisters with their first cousins once removed. Certain observations of Sjögren (1950), however, do not exclude the possibility of an occasional dominant transmission.

X. MONGOLISM

The principal signs of mongoloid idiocy (fig. 24) are the following: both physical and mental retardation; small stature; flat, round and mongoloid face; saddle nose; thick and fissured lower lip; large, scrotal tongue; reduction of

the anteroposterior diameter of the skull; absence of the frontal sinuses; abnormal development of bones of the face and skull; short and stocky limbs; soft and seborrheic skin; thick, smooth hair; obesity; genital insufficiency; muscular hypotonia and great looseness of the joints; very pronounced psychic disturbances.

FIG. 24—Mongolism.

In the eye one notes hypertelorism and narrow palpebral fissures which run obliquely downwards and inwards. There is frequently an epicanthus. The free edge of the upper eyelid is strongly arched. The stroma of the iris is very thin and presents a characteristic mottled appearance. There is often a convergent squint and nystagmus.

More than half of the patients with mongolism have cataract, which is sometimes evolutive. In a third of the cases a high degree of myopia is present.

Four types of ocular opacity are distinguished in these patients:

1. Arcuate opacities astride the equator of the fetal nucleus. The opacities, often thin and narrow, are sometimes fairly wide, thus occupying an important sector. Sometimes they constitute a true zonular cataract. They may be divided into two parts and separated by a thin layer of transparent fibers.

2. Opacities of the embryonic sutures.

3. Various congenital cataracts (polar, posterior).

4. Acquired opacities, similar to cerulean cataract (Pearce, Rankine and Ormond, 1910; Ormond, 1911-1912; van der Scheer, 1919; Vontobel, 1933; Bucklers, 1939).

In examining 52 mongols, Lowe (1949, 1950) found arcuate opacities eight times, sutural opacities fourteen times, a disc-shaped cataract once, a zonular cataract once, and a posterior polar cataract once — twenty-five congenital cataracts.

Skeller and Oster (1951) reported that in 32 mongols they found eight eyes with arcuate opacities, ten eyes with sutural opacities, and eleven eyes with various congenital cataracts; that is, 45 per cent of the eyes with congenital cataracts. They found 45 eyes with acquired opacities (70 per cent). Igersheimer and Mautner (1951) among 165 mongols found 8 per cent with congenital cataracts. Other authors also have reported congenital cataracts or opacities: Cassel (1917, 3 cases out of 60 mongols), van der Scheer (1919, 2 cases of zonular cataract out of 90 cases of mongoloid idiocy), Chotzen (1925), Elschnig (1932), Vogt (1933), Vidal Freyre et al. (1936), Scatena and Gallo (1941), Spitzer and Mann (1950).

The pathogenesis of mongolism is still a very controversial subject. Generally, advanced age of the mother at the moment of conception is invoked as the cause. There are, however, some arguments in favor of the intervention of a predisposing hereditary factor, transmitted with simple recessivity:

1. According to Schroeder (1939), the incidence is as high as 88 per cent in monozygote twins and only 8 per cent in dizygotes. Benda (1949) found an incidence in 7 cases out of 7 pairs of monozygote twins against 4 cases out of 25 pairs of dizygote twins; Gedda (1951), in 26 cases out of 28 pairs of monozygotes against 4 cases out of 60 pairs of dizygotes; Jervis (1953), in 20 cases out of 20 pairs of monozygotes against 4 cases out of 62 pairs of dizygotes; Allen and Kallmann (1956), in 3 cases out of 3 pairs of monozygotes against no case in 23 pairs of dizygotes.

Although cases of discordance have been reported in monozygote twins (Orel, 1931; Oster, 1953; Friedman, 1955; Van Beukering and Vervoorn, 1956), it may be said that concordance is the rule among monozygotes (nearly 100 per cent according to Allen and Kallmann) and the exception among dizygotes (4 per cent according to the same authority). We have had the opportunity to observe a pair of univitelline female twins, age 1.5 years, both of which presented a mongoloid idiocy with erythremia and albinism.

2. There exist familial cases, with affection of several members of the same sibship: Portius (1941) found 15 such in 460 mongolians; Böök and Reed (1950) observed 4 cases of mongolism among 271 first cousins of 63 mongolians; Turpin and Lejeune (1953) saw 4 mongolian brothers and sisters out of 7 members of a sibship; Oster (1956) found 30 familial cases out of 526.

Consanguinity of the parents, however, is not frequent: 1.2 per cent according to Portius (1941), and 0.57 per cent according to Oster (1956).

XI. SYNDERMATOTIC CONGENITAL CATARACTS

Certain cutaneous affections such as poikiloderma infantilis of Zinsser-Thomson (Rothmund's syndrome), congenital dyskeratosis (Schafer's syndrome), congenital ichthyosis, congenital atrophy of the skin (Siemens' syndrome) and patchy pigmentary dermatosis (Incontinentia pigmenti) are often accompanied by a congenital cataract.

Of course we are not speaking here of acquired syndermatotic cataracts (scleropoikiloderma of d'Arndt-Jaff, the syndrome of Werner, Besnier's prurigo).

Rothmund's Syndrome

Rothmund's and Werner's syndromes have been confused for a long time; it is only in recent years that the two affections have been separated in a precise manner.

Whereas the syndrome of Werner begins after puberty, that of Rothmund begins in the course of the first year of life, generally between the third and sixth months, by a cutaneous eruption of exanthematous type. It is relatively rare, as only about 25 cases are known.

It is characterized by three symptoms: dermatosis, cataract, and hypogenitalism.

1. Dermatosis. The eruptive and exudative lesions develop towards cicatrization and sclerosis to constitute a poi-

FIG. 25—Rothmund's syndrome (after Habermann and Fleck, 1955).

kiloderma of the infantile type of Zinsser-Thomson (fig. 25 and 26). Three kinds of lesions are distinguished:

a. Zones of atrophy and of sclerosis, giving to the palpating finger the impression of cigarette paper.

b. Telangiectases, the color of the lees of wine, which predominate in the immediate vicinity of the cicatricial zones.

c. Derangements of pigmentation: depigmented and hyperpigmented areas situated outside the atrophic zones and more especially on the trunk.

The cutaneous alterations are mainly found on the face, ears, forearms, hands and legs.

Disturbances of the phanera (hair, nails, etc.) are added to the cutaneous lesions:

a. Alterations of the pilary system: the hair falls out and becomes thin (alopecia or early baldness); the eyelashes and eyebrows are thin and sometimes absent.

b. Dystrophy of the nails; shortened, striated or atrophied nails, associated with an acromicria (small hands and fingers).

FIG. 26—Rothmund's syndrome (after Seefelder, 1935).

It is not uncommon to see dental anomalies (Bloch, 1926; Jackli 1939; Cole et al., 1945).

Finally, attention may be drawn to the nose, which is small, saddle-shaped and pale; the skin there is particularly atrophic and traversed by very fine telangiectases.

2. Cataract. This cataract of the endocrine type generally begins when the child is between the second and fourth years of age. It is always bilateral and evolves rapidly in a few weeks or months. It may be congenital and of the zonular type, as in the cases of Cole and his colleagues (1945).

Exceptionally, there is no opacity of the lens (Habermann and Fleck, 1955, 2 cases).

Degenerative lesions of the cornea have also been reported: Rothmund (1869) and Seefelder (1935); Strebel (1913), Bloch and Stauffer (1929); Schnyder (1935) and Maeder (1949).

3. Hypogenitalism. The endocrine disturbances which commence with the onset of puberty are generally reduced to a hypoplasia of the internal and external genital organs, with amenorrhoea in the woman and testicular atrophy in the man. Bloch and Stauffer (1929) observed an adiposo-genital syndrome. In the cases of Jackli (1939) and of Miescher (1941) there was a nanism.

Habermann and Fleck (1955) observed a disturbance of enchondral ossification, both epiphyseal and metaphyseal, in a brother and sister, both of them affected by the condition.

Heredity

The syndrome of Rothmund is much more frequent in women than in men. Out of the 24 cases collected by Maeder (1949), there were 19 women and 5 men; the difference is significant (X2: 7).

The affection is always observed within the same sibship (Rothmund, 1868, 3 sibships, with, respectively, 4 first cousins affected out of 7, 2 out of 6, and 2 out of 3; Siegrist-Lutz 1928, 2 first cousins affected out of 4; Seefelder, 1935, 3 first cousins affected out of 6; Cole et al., 1945, 2 first cousins out of 2, Maeder, 1949, 4 first cousins affected out of 10).

Consanguinity of the parents is, moreover, reported in 4 cases (Rothmund, 1868, 2 cases; Seefelder, 1935; Guillaumat and Maeder, 1949; Habermann and Fleck, 1955).

It may therefore be concluded that there is an autosomal recessivity.

Schafer's Syndrome

This syndrome consists of a congenital dyskeratosis associated with a congenital cataract. Depending on a dominant hereditary factor, it has the following characteristics:

1. A palmoplantar keratosis (fig. 27) with hyperhidrosis, a disseminated follicular hyperkeratosis of the skin, and a leukokeratosis of the buccal mucosa.

2. Small foci of cicatricial alopecia.

3. A pachyonychia: very thick nails resting on a tissue like the pith of a reed.

4. A retardation of development (microcephaly and nanism) with oligophrenia and hypogenitalism.

5. A congenital cataract.

Congenital Ichthyosis

Congenital ichthyosis or congenital ichthyoform erythrodermia of Brocq (1902) consists of a diffuse erythema more or less thick, in patches or sheets, diffuse or generalized. The hyperkeratosis especially involves the articular folds (fig. 27 and 28), the palms and the soles. Here, may be noticed, a simple dryness of the skin with fine desquamation, but also masses, cornified, yellowish or amber, villous and macerated or black and

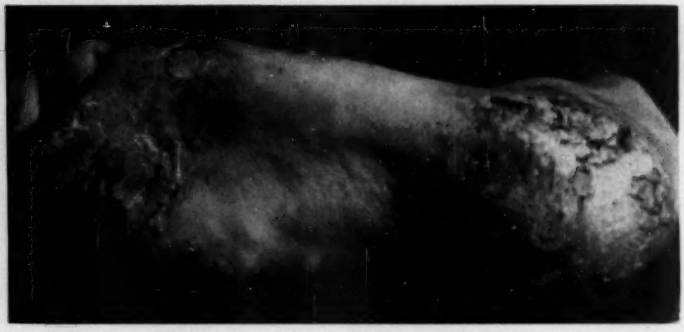

FIG. 27—Keratodermia of the soles (after Graciansky and Boulle, 1952).

dry, often arranged in linear series in the axillae, irregular and cut by deep fissures, painful, bleeding and malodorous on the palms and soles (Graciansky and Boulle, 1952).

Erythema is frequent, although not always present. It may be generalized, especially in the child, or localized to certain regions. It is easily seen through the rather thin, smooth, shining, cracked, and collodion-like hyperkeratosis of the face, and above all, in the form of a keen red edge a few millimeters wide, on the outer borders of the sheet of hyperkeratosis of the soles and palms. Hyperhidrosis and sebaceous secretion are usual, as well as the hyperepidermotropia, which is evidenced by the exaggerated growth of the nails and hairs. The affection, which presents a heredity that is generally recessive and often with consanguinity of the parents (Adrian, 1906; Siemens, 1929; Gottron, 1933; Cockayne, 1933), may be accompanied by a *congenital* cataract (Siemens, 1929).

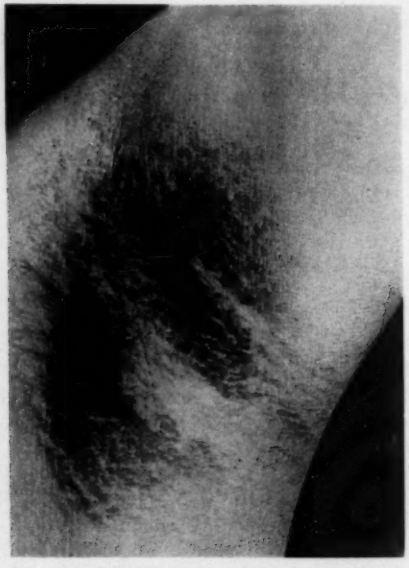

FIG. 28—Congenital ichthyoform hyperkeratosis (after Graciansky and Boulle, 1952).

Siemens' Syndrome

Siemens (1929) described the association of a congenital cataract and a congenital atrophy or hypoplasia of the skin (fine, somewhat transparent skin easily falling into folds, fragile, lacking fat, slightly shaggy and poor in sudoriparous glands).

The affection is hereditary and the transmission is simple recessive: six cases distributed among three related sibships.

Incontinentia Pigmenti

Franceschetti and Jadassohn (1954) have recently shown that two separate syndromes must be distinguished: (1) infantile pigmentary dermatosis in Bloch-Sulzberger patches and (2) reticulated infantile pigmentary dermatosis.

The latter affection, which is characterized by a slate-colored, cutaneous pigmentation of reticulated morphology, hypohidrosis or anhidrosis, a bad state of the teeth, and a palmoplantar keratosis, is not accompanied by ocular anomalies.

Pigmentary dermatosis in patches

This syndrome, described by Bloch (1925) and Sulzberger (1926), is generally congenital or appears in the course of the first week of life (in 63 per cent of cases), rarely in the course of the first year, and exceptionally after one year.

It is characterized by the following symptoms:

1. Slate-gray cutaneous pigmentation in patches, disposed in striae of wavy streaks and sometimes disappearing at the end of several years. Histologically, chromatophores are seen in the superficial layers of the derma with more or less complete absence of pigment in the epidermis. In two thirds of the cases the pigmentation is preceded by an acute inflammatory stage, which is often of the type of Duhring's dermatitis herpetiformis.

2. Ocular affections, generally of the malformation type, exist in a quarter of the cases: pseudoglioma (Haxthausen, 1945; Duke University, 1948; Uebel et al., 1950; Findlay, 1952; Bloch, Sulzberger, Franceschetti and Jadassohn, 1954; Kawamura, 1954; Watanabe, 1954; Scott et al., 1955); atrophy of the optic nerve (Moncorps - Seidlmayer, 1942; Seidlmayer-Höra, 1943; Gerard-Lapiere, 1959; Carney, 1959). Nystagmus has been reported (Haber, 1952), a strabismus (Lechleuthner-Siemens, 1929; Pierini et al., 1952), and a probable papilitis (Kühling 1949).

Fairly often a congenital cataract has been noted (Sulzberger, 1938; Jaramillo et al., 1948; Curth, 1949; Gasteiger, 1959; Ito, 1951).

3. In a quarter of the cases, dental anomalies have been reported (partial absence of the teeth, dental malformations, delay in dentition).

4. Also in a quarter of the cases, there is an alopecia of the pseudo-pelada type of Brocq.

Heredity. The first feature that catches the attention is the extraordinary predominance of the feminine sex: out of the 73 cases culled from the literature by Franceschetti and Jadassohn (1954), there are 70 women; and out of the 91 cases collected by Scott and his colleagues (1955), there were 86.

Four cases only were familial: two identical twins of Bardach (1925), two sisters of Curth (1949), two others of Kitamura and Fukishiro (1951), and two female first cousins reported by Scott and his colleagues (1955).

Consanguinity of the parents is reported only twice (Lapiere-van Bogaert-Gerard, 1947 and 1948; Kitamura and Fukishiro, 1951).

Nevertheless, it must be noted that ocular malformations have been observed in the parents of affected subjects (Sulzberger, 1938; Duke University, 1938; Franklin, 1952; Findlay, 1952; Scott et al., 1955), that sometimes an affection suspected of being incontinentia pigmenti has been reported in the ancestors (Sobel, 1948; Kitamura

and Fukishiro, 1951), that one may often find symptoms frequently associated with incontinentia pigmenti in other members of the family (pseudo-pelada and dental anomalies). Franceschetti and Jadassohn (1958) believe that there may be a dominant transmission with great variability of expressivity and of penetrance.

CONCLUSION

It is clear that if, in the presence of a congenital cataract whose etiology appears to us obscure, we take the trouble to make a general and systematic examination that will collect all the concomitant anomalies, we shall very often be able to refer it to one or another syndrome, and by this very fact attribute to it a genetic origin.

REFERENCES

I.

1. Albright, Fuller; Butler, Allan M., and Bloomberg, Esther: Rickets resistant to vitamin D therapy, Am. J. Dis. Child., 54:529-547, 1937.

2. Appelmans, M.; Michaels, J.; Walravens, A., and Doyen, N.: Les manifestations oculaires du syndrome de Lowe, Bull. Soc. belge opht, 116:311-321, 1957.

3. Bickel, H., and Thursby-Pelham, D. C.: Hyperamino-aciduria in Lignac-Fanconi disease, in galactosaemia and in an obscure syndrome, Arch. Dis. Child., 29: 224-231, 1954.

4. Debre, Robert; Marie, Julien; Cleret, F., and Messimy, R.: Rachitisme tardif coexistant avec une néphrite chronique et une glycosurie, Arch. Med. Enf., 37: 597-606, 1934.

5. Debre, R.; Royer, P.; Lestradet, H., and Straub, W.: L'insuffisance tubulaire congenitale avec arrieration mentale, cataracte et glaucome, Arch. franc. pediat., 12:337-348, 1955.

6. Dent, C. E., and Harris, H.: Hereditary forms of rickets and osteomalacia, J. Bone & Joint Surg., 38-B:204-226 (Feb.) 1956.

7. De Toni, G.: Remarks on the relations between renal rickets (renal dwarfism) and renal diabetes, Acta Paediat., 16: 479-484, 1933.

8. Fanconi, G.: Der frühinfantile nephrotisch-glykosurische Zwergwuchs mit hypophosphatämischer Rachitis, Jb. Kinderhk., 147:299-338 (Oct.) 1936.

9. Franceschetti, A.: Discussion de la communication de Streiff et collaborateurs, Ophthalmologica, 135:638-639, 1958.

10. Gérard-Lefebvre, Biserte, G., et al.: Étude clinique, génétique et biologique du syndrome de Lowe-Bickel, Pediatrie, 12:527-534, 1957.

11. Grislain, J. R.: Reflexions sur 4 cas de cystinose et 1 cas de syndrome de Lowe, Soc. Portug. de Pediatrie, October 1957.

12. Lignac, G. O. E.: Über Störung des Cystinstoffwechsels bei Kindern, Deutsches Arch. klin. Med., 145:139-150 (Oct.) 1924.

13. Lowe, Charles Upton; Terry, Mary, and MacLachlan, E. A.: Organic-aciduria, decreased renal ammonia production, hydrophthalmos, and mental retardation: a clinical entity, Am. J. Dis. Child., 83: 164-184 (Feb.) 1952.

14. Monnet, P.; Matray, and Etienne: Glaucome congénital et néphropathie, Pediatrie, 10:617-622, 1955.

15. Monnet, P.; Creyssel, R.; Matray, and Gilly, R.: Hypoplasie musculaire généralisée avec oedémes lymphangiectasiques. Syndrome de Bonnevie-Ullrich probable—Association avec une oculopathie de nature mal précisée et une nephropathie comportant une hyperamino-acidurie, Pediatrie, 10:655-657, 1955.

16. Schlesinger, B.: Cited by Dent and Harris.[6]

17. Streiff, E. B.; Straub, W., and Golay, L.: Les manifestations oculaires du syndrome de Lowe, Ophthalmologica, 135: 632-639, 1958.

II.

18. Alexander: Cited by Patry.[25]

19. Aubaret, E., and Guillot: Oxycephalie et cataracte zonulaire, Rev. d'oto-neuro-ophtal., 12:279-280 (April) 1934.

20. Falls, Harold F.: Developmental cataracts: results of surgical treatment in one hundred thirty-one cases, Arch. Ophth., 29:210-223,1943.

21. Gänsslen, M.: 2. Die hämolytische Konstitution. Konstitutionneller hämolytischer Ikterus (hämolytische Anämie Kugelzellenkrankheit.) In Handbuch der Erbbiologies des Menschen, Berlin, J. Springer, 1940, vol. 4, pt. 1, pp. 415-419.

22. Groenouw: Cited by Patry.[25]

23. Kraus: Cited by Patry.[25]

24. Lartigue, A.: Contribution à l'étude de la cataracte dans l'oxycéphalie, Paris, Thèse, 1939.

25. Patry, A.: Contribution à l'étude des lèsions oculaires dans les malformations crâniennes, spécialement dans l'oxycephalie, Paris, Thèse, 1905.

26. Renard, G., and LaPorte, P.: Les cataractes congénitales à cristaux, Arch. opht., 11:739-753, 1951.

27. Sourdille, G. P.; Eoche-Duval, and Gendron: Cataractes congénitales et oxycéphalie, Bull. Soc. opht. France, 51:186-187 (March) 1939.

28. Vest, M.: Rubeolenembryopathie als Ursache von Akrocephalosyndaktylie und Turmschädel, Ann. Paediat., 184:14-25, 1955.

29. Walsh, Frank Burton: Clinical Neuro-Ophthalmology, Baltimore, Williams and Wilkins, 1947.

III.

30. Appenzeller, G. F. A.: Beitrag zur Lehre von der Erblichkeit der grauen Stars, Tübingen, Thèse, 1884.

31. Ginestous, E., and Lavie, E.: Cataracte congénitale double et syndactylie, cited from Ginestous, E.: Ophtalmologie infantile, Paris, G. Doin, 1922.

32. Karsh, Johannes: Zur Klinik und Erbbiologie des angeborenen Stars, ins besondere des Schichtstars, Ztschr. f. Augenh., 92:322-335, 1937.

33. Koby, F. Ed.: Cataracte familiale d'un type particulier, se transmettant apparemment suivant le mode dominant, Arch. opht., 40:492-503, 1923.

34. Manson, J. S.: Hereditary lamellar cataract and digital deformity in the same pedigree, Tr. Ophth. Soc. U. Kingdom, 32:45-53, 1912.

35. Rieger, H.: Erbfragen in der Augenheilkunde, von Graefes Arch. Ophth., 143:277-299, 1941.

36. Rollet and Genet: Cataracte congénitale familiale, Lyon Med., 120:1423-1425, 1913; Rev. gen. d.'opht., 32:418, 1913.

IV.

37. Armaly, Mansour F.: Ocular involvement in chondrodystrophia calcificans congenita punctata, A.M.A. Arch. Ophth., 57:491-502 (April) 1957.

38. Bateman, Donald: Two cases, and specimens from a third case, of punctate epiphyseal dysplasia, Proc. Roy. Soc. Med., 29(pt. 1):745-746, 1935-36.

39. Bedford, H. Berrey, and Kimball, Cecil H.: Chondrodystrophia calcificans congenita, J. Pediat., 42:474-477, 1953.

40. Bergstedt, Jens, and Karlén, Karl-Henrik: Chondroangiopathia calcarea s. punctata (chondrodystrophia calcificans congenita), Monatsschr. Kinderh., 102: 347-349, 1954.

41. Bloxsom, Allan, and Johnston, Robert A.: Calcinosis universalis with unusual features, Am. J. Dis. Child., 56:103-109, 1938.

42. Borovsky, Maxwell P., and Arendt, Julian: Chondodystrophia calcificans congenita, J. Pediat., 24:558-573, 1944.

43. Bossi, Renzo, and Pisani, Giovanni: Stippled epiphyses in a newborn infant without micromelia, Brit. J. Radiol., 27: 449-452 (Aug.) 1954.

44. Brychnac., V.: Chondrodystrophia calcificans congenita, Acta, Chir. Orthop. Traum. Cesch., 23:328-331 (Nov.) 1956.

45. Burckhardt, Edward: Ein Fall von chondrodystrophia fetalis calcarea, Schweiz. med. Wchnschr., 68:330-334, 1938.

46. Buxton, J. D.: A dwarf with stippled epiphyses, Proc. Roy. Soc. Med., 23 (pt. 2):1329-1331, 1930.

47. Contradi, Erich: Vorzeitiges Auftreten von Knochen- und eigenartigen, Verkalkungskernen bei Chondrodystrophia fötalis hypoplastica. Histologische und Röntgenuntersuchungen, Jahrb. Kinderh., 80:86-97, 1914.

48. Coughlin, Edward J., Jr.; Guare, Howard T., and Moskovitz, Abraham J.: Chondrodystrophia calcificans congenita; case reports with autopsy findings, J. Bone & Joint Surg., 32-A:938-942 (Oct.) 1950 .

49. Dietrich: Festschr. Med. Akademie, Köln, 1915, p. 122; Hdb. Spez. Path., Henke-Lubarsch, 1:9, 1929.

50. Fairbank, H.A.T.: General diseases of the skeleton, Brit. J. Surg., 15:120-142 (July) 1927 .

51. ———: Generalized diseases of the skeleton, Proc. Roy. Soc. Med., 28 (pt. 2): 1611-1619 (Oct.) 1935.

52. ———: Displasia epiphysialis punctata, J. Bone & Joint Surg., 20:114, 1938.

53. ———: Dysplasia epiphysialis punctata. Synonyms—stippled epiphyses, chondrodystrophia calcificans congenita (Hünermann), J. Bone & Joint Surg., 31-B:114-117 (Feb.) 1949.

54. ———: Dysplasie epiphysialis punctata, J. Bone & Joint Surg., 33-B:461, 1951.

55. Fanconi, G.: Ueber generalisierte Knochenerkrankungen im Kindesalter, Helvet. paediat. acta, 2:3-32, 1947.

56. Figueroa, J. M., and Rangel, L.: Un caso de condrodistrofia calcificante congenita, Proc. Med. Hosp. Inf., 13:819-823, 1936.

57. Ford, G. David; Schneider, Martin, and Brandon, James R.: Congenital stippled epiphyses, Pediatrics, 8:380-392 (Sept.) 1951.

58. Frank, W.W., and Denny, M.B.: Dysplasia epiphysialis punctata; report of a case and review of literature, J. Bone & Joint Surg., 36-B:118-122 (Feb.) 1954.

59. Fraser, F. Clarke, and Scriver, Jessie Boyd: A hereditary factor in chondrodystrophia calcificans congenita, New England J. Med., 250:272-277 (Feb. 18) 1954.

60. Geyman, Milton J.: An unusual manifestation of epiphyseal and joint pathology in a new-born infant, Am. J. Roentgenol., 26:868-870 (Dec.) 1931.

61. Grebe, H.: Chondrodysplasia-chondromalazia, Zentralbl. allg. Path., 88:403-409 (May 5) 1952.

62. Gruber, G. B.: Chondodrodystrophia fetalis. In Schwalbe, Ernst: Die Morphologie der Missbildungen des Menschen und der Tiere, Jena, G. Fischer, 1937, vol. 3, pp. 333-361.

63. Gubern, Salisachs L.; Claret, Corominas I., and Domenech, Claros A.: Chondrodistrofia calcificans congenita, Acta pediat. espan., 15:197-202 (March) 1957.

64. Harris, H. A.: Bone Growth in Health and Disease, London, Oxford University Press, 1933.

65. Hässler, E., and Schallock, G.: Chondrodystrophia calcificans, Monatschr. Kinderh., 82:133-157, 1940.

66. Haynes, Earl R., and Wagner, Wm. F.: Chondroangiopathia calcarea punctata, Radiology, 57:547-550 (Oct.) 1951.

67. Hilliard, Cedric: A case of chondro-osseous dystrophy with punctate epiphyseal dysplasia, Brit. J. Radiol., 16:144-146 (May) 1943.

68. Hünermann, Carl: Chondrodystrophia calcificans congenita als abortive Form der Chondrodystrophie, Ztschr. Kinderh., 51:1-19, 1931.

69. Hutchison, James H., and McDonald, A. M.: Chronic acidosis in infants due to renal tubular deficiency: its association with metastatic calcification, Acta Paediat., 40:371-400 (Sept.) 1951.

70. Huurman: A case of calcifying chondrodystrophy, Nederl. tijdschr. Verlosk. en gynaec., 53:313-316, 1953.

71. Jeune, M.; Larbre, F.; Carron, R., and Couette, I.: La maladie congenitale des épiphyses pointillees ou calcinose foetale épiphysaire chondrodystrophiante, Arch. franç. pediat. 10:914-942, 1953.

72. Jolly, Hugh: Punctate epiphyseal dysplasia, Proc. Roy. Soc. Med., 44:227, 1951.

73. Jorup, Sigvard: Fall von Chondrodystrophia congenita calcificans, Acta Radiol., 25:580-586, 1944.

74. Kampf, E.: Chondrodystrophia calcificans congenita, Ztschr. Kinderh., 61:124-126, 1939.

75. Karlen, A. G., and Cameron, J. A. P.: Dysplasia epiphysialis puncta. Report of a case with discussion, J. Bone & Joint Surg., 39:293-301 (May) 1957.

76. Karlén, K. H.: A case of congenital chondrodystrophia calcificans, Acta paediat., 42:391, 1953.

77. Käser, H.: Chondrodysplasia calcificans congenita, Schweiz. med. Wchnschr., 87: 676-680, 1957.

78. Kass, A.: Chondrodystrophia congenita calcificans punctata, Acta paediat., 44: 134, 1955.

79. Kremens, V., et al.: Congenital calcific chondrodystrophy, J. Albert Einstein M. Center, 3:137-144, 1955.

80. Kwerch, H.: Zur Kenntnis der Sonderformen der Chondrodysplasia (Chondrodystrophia) foetalis im besonderen der Chondrodysplasia calcificans congenita, Oesterr. Ztschr. Kinderh., 4:165-180, 1950.

81. de Lange, Cornelia, and Janssen, Tieline: Chondrodystrophia calcificans congenita als Onderdeel van een Reeks Aangeboren Afwijkingen [Chondrodystrophia calcificans congenita of infant in association with other abnormalities], Maandschr. kindergeneesk., 17:67-74, 1949.

82. Licht, J., and Jesiotr, M.: Chondroangiopathia calcarea seu punctata [Chondrodystrophia calcificans congenita]: an atypical, stationary form of the disease, Am. J. Roentgenol., 78:492-498 (Sept.) 1957.

83. Liess, G.: Zur Chondrodystrophia calcificans congenita, Fortschr. Geb. Röntgenstrahlen., 81:61-65 (July) 1954.

84. Lightwood, R. C.: Congenital deformities with stippled epiphyses and congenital cataract, Proc. Roy. Soc. Med., 24: 564-566 (pt. I) 1930-31.

85. Lund, E.: Metaphyseal dyscrasia, Proc. Roy. Soc. Med., 36:381, 1943.

86. Maitland, D. G.: Punctate epiphyseal dysplasia occuring in two members of the same family, Brit. J. Radiol., 12:91-93 (Feb.) 1939.

87. McCullough, J. A. L., and Sutherland, Charles G.: Epiphyseal dysplasia punctcularis (stippled epiphyses). Report of a case not associated with hypothyroidism, Radiology, 34:131-135 (Feb.) 1940.

88. Paufique, L., and Étienne, R.: Cataracte de la calcinose foetale polyepiphysaire chondrodystrophiante (maladies des epiphyses pointillées) cataracte de la galactosémie, Bull. Soc. opht. France, 67: 42-44, 1954.

89. Paul, Lester W.: Punctate epiphyseal dysplasia (chondrodystrophia calcificans congenita): report of case with nine year period of observation, Am. J. Roentgenol., 71:941-946 (June) 1954.

90. Phillips, L. I.: Chondrodystrophia calcificans congenita, New Zealand M. J., 56:22-27, 1957.

91. Raap, Gerard: Chondrodystrophia calcificans congenita, Am. J. Roentgenol., 49:77-82 (Jan.) 1943.

92. Reilly, William Anthony, and Smyth, Francis Scott: Stippled epiphysis with congenital hypothyroidism (cretinoid epiphyseal dysgenesis) Am. J. Roentgenol., 40:675-681 (Nov.) 1938.

93. Resnick, Eber: Epiphyseal dysplasia punctata in a mother and identical male twins, J. Bone & Joint Surg., 25:461-468 (April) 1943.

94. Sardi, A.: Un caso di condrodistrofia punctata congenita, Minerve ortop., 7:8-11, 1956.

95. Savignac, Eugene M.: Chondrodystrophia calcificans congenita, Radiology, 58:415-420 (March) 1952.

96. Schönenberg, H., and Schallock, G.: Zur Kenntnis der Chondrodysplasia calcificans congenita und ihrer Beziehungen zur Chondrodysplasia foetalis, Ann. paediat., 180:129-162, 1953.

97. Scott, L. G.: Dysplasia epiphysealis punctata, Proc. Roy. Soc. Med., 45: 453, 1952.

98. Sears, W. Gordon: Dwarfism with stippled epiphyses, Brit. J. Child. Dis., 28:290-295 (Oct.-Dec.) 1931.

99. Selakovich, Walter G., and White, J. Warren: Chondrodystrophia calcificans

congenita; report of a case, J. Bone & Joint Surg., 37-A:1271-1277 (Dec.) 1955.

100. Sheach, Jean M., and Middlemiss, J. H.: Dysplasia epiphysialis punctata, Brit. J. Radiol., 29:111-113 (Feb.) 1956.

101. Stopp, H.: Kasuistischer Beitrag zur Chondrodystrophia calcificans congenita. [Case report on congenital dyschondroplasia calcificans], Ztschr. Arztl. Fortbild., 51:196-199 (March) 1957.

102. Straight, B.: Chondrodystrophia calcificans congenita, Clin. Proc. Child. Hosp., 10:182-188, 1954.

103. Stüve, A.: Zur Chondrodystrophia calcificans congenita, Kinderärztl. Praxis, 21:314-317 (July) 1953.

104. Swoboda, W.: Beitrag zur Chondrodystrophia calcificans congenita, Ann. paediat., 175:322-342, 1950.

105. ———: Chondrodystrophia calcificans congenita, Monatsschr. Kinderh., 100: 444-447 (Oct.) 1952.

106. Tisdall, Frederick F., and Erb, I. H.: Report of two cases with unusual calcareous deposits, Am. J. Dis. Child., 27:28-38, 1924.

107. de Toni, G. and Papio, F.: La chondrodystrofia congenita calcificante: primo contributo casistico italiano, Policlinico infantile, 16:3-10, 1948.

108. Vinke, Theodore H., and Duffy, F. Paul: Chondrodystrophia calcificans congenita. Report of two cases, J. Bone & Joint Surg., 29:509-514 (April) 1947.

109. Vychytil, Otto: Besondere Form der Chondrodystrophie (Rare form of chondrodystrophy), Casop. lék. cesk., 70: 1785-1791 (Dec. 18) 1931; abstr. Zbl. Kinderh., 26:611, 1931.

110. Weber, A.: Zur Frage der Chondrodysplasia calcificans congenita, Helvet. paediat. acta, 13:228-238 (Aug.) 1958.

111. Wilkins, Lawson: Epiphysial dysgenesis associated with hypothyroidism, Am. J. Dis. Child., 61:13-34 (Jan.) 1941.

112. Wiskott, Alfred: Ueber neuere Behandlungsverfahren der allgemeinen Rachitis-bekämpfung. III. Mitteilung: Bestrahltes Ergosterin gegen Rachitis, München Med. Wchnschr., 76:1430-1433 (Aug. 23) 1929.

113. Baumann, T.: Discussion du rapport de Rossi E.: Le tableau clinique de status Bonnevie-Ullrich d'après les cas du Kinderspital Zurich, Ann. paediat., 165: 158-160, 1945.

114. Beyer, P.; Lausecker, V.; Levy, J. M., and Bartolo, R.: Une nouvelle observation d'arthrogryposis multiplex et de pterygium, Arch. franç. pédiat., 13:286-293, 1956.

115. Bonnevie, Kristine: Embryological analysis of gene manifestation in Little and Bagg's abnormal mouse tribe, J. Exper. Zool., 67:443-520, 1934.

116. ———: Vererbbare Missbildungen und Bewegungsstörungen embryonale Gehirnanomalien zuruckfuhrbar, Erbartz, 2:145-150, 1935.

117. Cotterman, C. W.: Abstracts of papers presented at the 1948 meetings of the Genetics Society of America, Washington, D. C., September 11-13, 1948, Genetics, 33:607-608, 1948.

118. Cotterman, C. W., and Falls, Harold F.: Unilateral developmental anomalies in sisters (status Bonnevie-Ullrich), Am. J. Human Genet., 2:203-213 (Dec.) 1949.

119. Foucar, H. O.: Pterygium colli and allied conditions, Canad. M. A. J., 59: 251-255 (Sept.) 1948.

120. Kieser, W.: Ztschr. menschl. Vererb.-u. Konstitutionslehre, 23:593, 1939.

121. Liebenam, L.: Zwillingspathologische Untersuchungen aus dem Gebiet der Anomalien der Korperform: Partieller Riesenwuchs; Angeborener Pectoralisdefekt; Dysostosis cleidocranialis; Dysostosis craniofacialis, Ztschr. menschl. Vererb.- u. Konstitutionslehre, 22:373-417, 1938.

122. Ostertag, Manfred, and Spaich, Dieter: Über die Pterygiumkrankheit und ihr diskordantes Vorkommen bei einem eineiigen Zwillingspaar, Deutsche Ztschr. Nervenh., 141:1-8 (July) 1936.

123. Rossi, Ettore: Le tableau clinique due status Bonnevie-Ullrich d'après les cas du Kinderspital de Zürich, Helvet. paediat. acta, 1(Suppl. 1-3):134-146, 1945.

124. Rossi, Ettore, and Caflisch, A.: Le syndrome du pterygium. Status Bonnevie-Ullrich, Helvet. paediat. acta, 2:98-102, 1947.

125. Rossi, Ettore, and Howald, Ernst: Über die Erblichkeit der Status Bonnevie-Ullrich, Helvet. paediat. acta, 2(Suppl. 4): 98-102, 1947.

126. Teveli, L.: Orv. Letil., 1:92-94, 1930.

127. Turner, H. H.: A syndrome of infantilism, congenital webbed neck, and cubitus valgus, Endocrinology, 23:566-574 (Nov.) 1938.

128. Ullrich, O.: Über typische Kombinationsbilder multiplex Abortungen, Ztschr. Kinderh., 49:271-276 (Feb. 28) 1930.

129. ———: Angeborene Muskeldefekte und angeborene Beweglichkeitsstörungen im Gehirnnervenbereich, Hdb. Neurol., Berlin, J. Springer, 1936, vol. 16, pp. 139-181.

130. ———: Turner's syndrome and status Bonnevie-Ullrich: a synthesis of animal phenogenetics and clinical observations on a typical complex of developmental anomalies, Am. J. Human Genet., 1:179-202 (Dec.) 1949.

131. ———: Der Status Bonnevie-Ullrich im Rahmen anderer "Dyscranio-Dysphalangien", Ergebn. inn. Med. u. Kinderh., 2:412-466, 1951.

132. Wertheman, and Reiniger: Odonto-Stomatologie, 2:267, 1949.

VI.

133. Aubry, M.: Variété singulière d'alopécie congénitale; alopécie suturale, Ann. dermat. et syph., 4:899-900, 1893.

134. Bergmeister, R.: Ein Fall von beiderseitiger kongenitaler Katarakt bei angeborener Schädelanomalie, Beitr. Augenh., 79:1-19, 1911.

135. Blodi, Frederick C.: Developmental anomalies of the skull affecting the eye, A.M.A. Arch. Ophth., 57:593-610 (April) 1957.

136. Cockayne, E. A.: Case reports: Dwarfism with retinal atrophy and deafness, Arch. Dis. Childhood, 11:1-8, 1936; 21: 52-54, 1946.

137. Franceschetti, A., and Klein, D.: The mandibulo-facial dysostosis: a new hereditary syndrome, Acta ophth., 27:143-224, 1949.

138. François, J.: Un nouveau syndrome. Dyscéphalie avec tête d'oiseau et anomalies dentaires, nanisme, hypotrichose, atrophie cutanée, microphtalmie et cataracte congénitale, Boll. ocul., 37:161-194 (March) 1958.

139. François, J., and Haustrate, L.: Anomalies colobomateuses du globe oculaire et syndrome du premier arc, Ann. ocul., 187:340-368, 1954.

140. Gilford, Hastings: Ateleiosis and progeria: continuous youth and premature old age, Brit. M. J., 2:914-918 (Oct. 8) 1904.

141. Gregersen, Eilif: Ocular abnormalities in progeria, Acta Ophth., 34:347-354, 1956.

142. Gregory, Irene D. R.: Congenital ectodermal dysplasia, Brit. J. Ophth., 39: 44-47, 1955.

143. Hallermann, W.: Vogelgesicht und cataracta congenita, Klin. Monatsbl. Augenh., 113:315-318, 1948.

144. ———: Über die Korrelation der Augen und Kieferentwicklung, Deutsche ophth. ges., 57:99-103, 1951.

145. Larmande, A.: Personal communication, 1959.

146. Leffertstra, L. J.: A case of dyscephalia with congenital cataract and hypotrichosis, Ophthalmologica, 132:204-206 (Sept.) 1956.

147. Ludwig, A., and Korting, G.: Vogt-Koyanagi-ähnliches Syndrom und mandibulofaciale Dysostosis (Franceschetti-Zwahlen). Arch. dermat. u. syph., 190: 307-316, 1950.

148. Marchesani, Oswald: Über Beziehungen zwischen Wachstum und Nervesegmenten, Deutsche ophth. ges., 55:34-38, 1949.

149. Marie, P., and Sainton, R.: Bull. et mem. Soc. med. hop. Paris, 14:706, 1897.

150. Moehlig, Robert C.: Progeria with nanism and congenital cataracts in a five year old child, J.A.M.A., 132:640-642 (Nov. 16) 1946.

151. Neill, Catherine A., and Dingwall, Mary M.: A syndrome resembling progeria: a review of two cases, Arch. Dis. Childhood, 25:213-221 (Sept.) 1950.

152. Nizetic, Branko: Dysmorphie mandibulo-faciale et status dysraphicus, Ophthalmologica, 127:1-13 (Jan.) 1954.

153. Norbis, A. L.: El ojo en las dismorfias mesodermicas, Arch. oftal. Buenos Aires, 27:302-315 (July); 364-375 (Aug.); 400-420 (Sept.) 1952.

154. Pau, Hans: Vogelgesicht mit Permeabilitätskatarakt als Folge trophischer Störungen, Klin. Monatsbl. Augenh., 117:529-532, 1950.

155. Ponte, F.: Viso a "Testa d'uccello" e cataratta congenita, Gior. ital. oftal., 11: 401-419 (Sept.-Oct.) 1958.

156. Scheutauer, G.: Allg. Wien. Med., 16: 293, 1871.

157. Schondel, Annie: Two cases of progeria complicated by microphthalmus, Acta paediat., 30:286-304, 1943.

158. Streiff, E. B.: Dysmorphie mandibulofaciale (tête d'oiseau) et altérations oculaires, Ophthalmologica, 120:79-83, 1950.

159. Ullrich, O., and Fremerey-Dohna, H.: Dyskephalie mit Cataracta congenita und Hypotrichose als typischer merkmalskomplex. Ophthalmologica, 125:73-90; 144-154 (Feb.) 1953.

160. Waardenburg, P. J.: Personal communication, 1958.

161. Walsh, Frank Burton: Clinical Neuro-Ophthalmology, ed. 2, Baltimore, Williams & Wilkins, 1957, p. 646.

162. Weyers, Helmut: Zur Dyscephalie mit Catarakta congenita und Hypotrichose, Ztschr. Kinderh., 74:468-483 (Jan.) 1954.

VII.

163. Bernard, Jean; Boiron, M., and Estager, J.: Une grande famille hémolytique; treize cas de maladie de Minkowski-Chauffard observés dans la même fratrie, Semaine hop. Paris, 28:3741-3744 (Dec. 14) 1952.

164. Campbell, J. M. H., and Warner, E. C.: Heredity of acholuric jaundice, Quart. J. Med., 19:333-355 (April) 1926.

165. Culbreath, P. H.: Synopsis of congenital hemolytic jaundice with preliminary report of two cases in identical twins, J. South Carolina M.A., 33:238-240 (Oct.) 1937.

166. Debré, Robert, et al: Maladie hémolytique chez deux jumeaux avec des déformations cranio-faciale et linguale, Bull. Soc. hop. Paris, 53:1465-1472 (Nov. 26) 1937.

167. Debré, Robert, et al: Étude de deux couples de jumeaux monozygotes atteints de maladie hêmolytique, Semaine hôp. Paris, 21:36-39, 1945.

168. Gänsslen, M.: Handbuch der Erbbiologies des Menschen, Berlin, J. Springer, 1940, vol. 4, pt. 1, p. 410.

169. Gasser, C.: Die hämolytischen Syndrome in Kindesalter, Stuttgart, Thieme, 1951.

170. Race, R. R.: On the inheritance and linkage relations of acholuric jaundice, Ann. Eugenics, 11:365-384, 1942.

171. Young, Lawrence E.; Izzo, Mary Jane, and Platzer, Richard R.: Hereditary spherocytosis: I. Clinical, hematologic and genetic features in 28 cases, with particular reference to the osmotic and mechanical fragility of incubated erythrocytes; II. Observations on the role of the spleen, Blood, 6:1073-1113, 1951.

VIII.

172. Aten, A. H.: Die Resultate von Operationen wegen angeborener Katarakt, Ophthalmologica, 102:120-121, 1941.

173. Bagley, Cecil H.: Congenital cataracts. A survey of the various types of operation, Am. J. Ophth., 32:411-419 (March) 1949.

174. Cordes, Frederick C.: Evaluation of the surgery of congenital cataracts, A.M.A. Arch. Ophth., 46:132-144 (Aug.) 1951.

175. Dufour, R.; Jequier, M.; Cuendet, J. F., and Michels, V.: Cataracte et oligophrenie, Bull. Soc. opht. France, 67:318-324, 1954.

176. Euziere, J.; Lafon, R., and Cazaban, A.: A propos du complexe du oligophreniemalformations oculaires, Vie Méd. Paris, 32:74-77, 1951.

177. Falls, Harold: Developmental cataracts: results of surgical treatment in one hun-

dred and thirty-one cases, Arch. Ophth., 29:210-223, 1943.

178. Franceschetti, A.: Discussion of Dufour, Jequier, Cuendet et Michels[175], Bull. Soc. opht. France, 67:324-327, 1954.

179. Franceschetti, A., and Brugger, C.: Über das kombinierte Auftreten von endogenem Schwachsinn und angeborenem Star, Schweiz. med. Wchnschr., 74:255-258, 1944.

180. Guillaumat, L.; Surugue, P., and Chabat, H.: Syndrome héréditaire de Sjögren: oligophrénie avec cataracte. [The hereditary syndrome of Sjögren: mental retardation with cataract], Bull. Soc. opht. Paris, 19:244-246 (May-June) 1948.

181. Gursdorf, C.; Hecaen, H., and Nau-Massonnet, M.: A propos de la maladie de Sjögren, Ann. Med.-Psychol., 110: 65-67 (Jan.) 1952.

182. Heuyer, G.; Bernard, and Leconte: Amyotrophie familiale chez deux frères et idiotie avec amaurose par cataracte congénitale chez deux frères: considérations communes, Zbl. ges. Ophth., 46: 235-236, 1941.

183. Heuyer, G., et al: Oligophrenie et cataracte, Arch. franç. pediat., 4:638-639 (Nov. 18) 1947.

184. Hofe, K. von: Beitrag zur Frage der Berufsfähigkeit derjenigen die in der Jugend wegen angeborenen Stars operiert wurden, Arch. f. Augenh., 110:34-38, 1936.

185. Kirman, Brian H.: Familial cataract and mental deficiency. Lancet, 1:694-696 (April 5) 1952.

186. Krause, Arlington C.: Congenital encephalo-ophthalmic dysplasia, Arch. Ophth., 36:387-444 (Oct.) 1946.

187. Pfändler, U.: Une souche de Vallon de Saint-Imier (Suisse), manifestant par consanguinité, la transmission récessive de malformations oculaires multiples (ectopie du cristallin, kératocône, cataracte congénitale, atrophie du nerf optique), associées à l'oligophrénie. Manifestation d'une achromatopsie totale dans une branche collatérale, Ophthalmologica, 119:105-113, 1950.

188. Rochat: Discussion of article by Aten, A. H.[172], Ophthalmologica, 102:121, 1941.

189. Schachter, M.: Ophthalmopathies congénitales et test de Rorschach, Acta neurol. et psychiat. belg., 50:151-156, 1950; Cataracte congénitale et débilité mentale (à propos de cinq observations), Pediatrie, 39:885-892, 1950.

190. Schachter, M., and Ourgaud, A. G.: Trois observations d'aniridie. (complexe ophthalmo-oligophrénique congénital, sporadique ou hérédo-familial), Arch. opht., 8:382-389, 1948.

191. Schappert-Kimmijser, J.: De betekenis van de congenitale cataract als oorsaak van slechtzienheid en blindheid. Personal communication, 1958.

192. Sjögren, T.: Klinische und Vererbungsmedizinische Untersuchungen über Oligophrenie mit kongenitaler Katarakt, Ztschr. ges. Neurol. Psychiat., 152:263-292, 1935.

193. Vignier, Christiane: Syndrome cataracte-oligophrenie de Sjögren, Paris, These, 1952.

194. Waardenburg, P. J.: Cataracta congenita, Genetica, 1:233, 1919.

IX.

195. Dufour, R.; Jequier, M.; Cuendet, J. F., and Michels, V.: Cataracte et oligophrénie, Bull. Soc. opht. France, 67:318-324, 1954.

196. Franceschetti, A.; Marty, F., and Klein, D.: Un syndrome rare: hérédoataxie avec cataracte congénitale et retard mental, Confinia Neurol., 16:271-275, 1956.

197. Garland, H., and Moorhouse, D.: An extremely rare recessive hereditary syndrome including cerebellar ataxia, oligophrenia, cataract and other features, J. Neurol. Neurosurg. & Psychiat., 16: 110-116 (May) 1953.

198. Marinesco, G.; Draganesco, S., and Vasiliu, D.: Nouvelle maladie familiale caractérisée par une cataracte congenitale et un arrêt du developpement somato-neuro-psychique, Encephale, 26:97-109 (Feb.) 1931.

199. Richards, B. W.: Cataracts, ataxia and mental deficiency: a report of two cases, J. Ment. Sc., 96:537-539, 1950.

200. Sjögren, Torsten: Hereditary congenital spinocerebellar ataxia combined with congenital cataract and oligophrenia, Ac-

ta psychiat. et neurol. (Suppl.) 46:286-289, 1947.

201. ———: Hereditary congenital spinocerebellar ataxia accompanied by congenital cataract and oligophrenia: a genetic and clinical investigation, Confinia neurol., 10:293-308, 1950.

X.

202. Allen, G., and Kallmann, F. J.: Mongolism in twin sibships, First International Congress Human Genetics, 1956, pp. 843-851.

203. Benda, Clemens Ernst: Mongolism and Cretinism; A Study of the Clinical Manifestations and the General Pathology of Pituitary and Thyroid Deficiency, New York, Grune & Stratton, 1946.

204. Beukering, J. A. van, and Vervoorn, J. D.: A case of uniovular twins of which one child was normal and the other had the syndrome of mongolism, Acta Genet. Med. et Gemell., 5:113-114, 1956.

205. Böök, J. A., and Reed, S. C.: Empiric risk figures in mongolism, J.A.M.A., 143:730-732 (June 24) 1950.

206. Bücklers, M.: Ueber Katarakt bei Mongolismus, Klin. Monatsbl. Augenh., 102: 144, 1939.

207. Cassel: Ueber Missbildungen am Herzen und an den Augen beim Mongolismus der Kinder, Klin. Wchnschr., 54:159-162, 1917.

208. Elschnig, A.: Starbildung bei mongoloider Idiotie, Klin. med., 1:217 (Feb.) 1932.

209. Friedman, Abraham: Mongolism in twins: its bearing upon the question of the etiology of mongolism, A.M.A. J. Dis. Child., 90:43-50, 1955.

210. Gedda, L.: Studio dei gemelli, Orizzonte medico, Roma, 1951.

211. Igersheimer, Joseph, and Mautner, Hans: About changes of the crystalline lens in mongoloids, Am. J. Ment. Deficiency, 55:370-376, 1951.

212. ———: About the significance of lenticular changes in mongolism, Ann. paediat., 181:34-44, 1953.

213. Jervis, George A.: A note on the etiology of mongolism, Quart. Rev. Pediat., 8:126-130 (Aug.) 1953.

214. Lowe, Ronald F.: The eyes in mongolism, Brit. J. Ophth., 33:131-174 (March) 1949.

215. ———: The arcuate lens opacities of mongolism and cataracts of similar appearance, Brit. J. Ophth., 34:484-488, 1950.

216. Orel, Herbert: Mongolismus bei Zwillingskindern, Ztschr. Kinderh., 51:31-38, 1931.

217. Ormond, Arthur W.: Notes on the ophthalmic condition of forty-two mongolian imbeciles, Tr. Ophth. Soc. U. Kingdom, 32:69-76, 1912.

218. Oster, J.: Mongolism. Opera ex Domo Biol., Herid. Human Univers. Halniensis, 52:1953; Dan. Med. Bull., 3:158, 1956.

219. Pearce, F. H.; Rankine, R., and Ormond, A. W.: Notes on twenty-eight cases of mongolian imbeciles: with special reference to their ocular condition, Brit. M. J., 2:186-190 (July 23) 1910.

220. Portius, W.: Fortschr. Erbpath. Rassenhyg., 5:194, 1941.

221. Scatena, A., and Gallo, L. A.: Congenital cataract in a mongoloid case, Rev. Soc. Pediat. de Rosario, 6:37-43, 1941.

222. Scheer, W. M. van der: Cataracta lentis bei mongoloider Idiotie, Klin. Monatsbl. Augenh., 62:155-170, 1919.

223. Schroeder, Hein: Die Sippschaft der mongoloiden Idiotie, Ztschr. ges. Neurol. u. Psychiat., 93:537-538, 1939.

224. Skeller, Erik, and Oster, Jakob: Eye symptoms in mongolism, Acta Ophth., 29:149-161, 1951.

225. Spitzer, Richard, and Mann, Ida: Congenital malformations in the teeth and eyes in mental defectives, J. Ment. Sc., 96:681-709 (July) 1950.

226. Turpin, R., and Lejeune, J.: Etude d'une famille comportant quatre freres et soeurs mongoliens, Semaine hop. Paris, 29:3979-3984 (Dec. 14) 1953.

227. Vidal Freyre, A., and Moret, R. L.: Mongolisme et cataracte congenitale chez

un syphilitique congenital, Arch. argent. de Pediat,. 7:831-837, 1936.

228. Vontobel, W.: Über Linsen- und Hornhautuntersuchungen an mongoloiden Idioten, von Graefes Arch. Ophth., 130: 325-338, 1933.

XI.

229. Adrian: Die Rolle der Consanguinität der Eltern in der Aetiologie einiger Dermatosen der Nachkommen, Dermat. Centralbl., 9:258-368, 1906.

230. Bardach, Martha: Systematisierte Naevusbildungen bei einem eineiigen Zwillingspaar. Ein Beitrag zur Naevusätiologie, Ztschr. Kinderh., 39:542-550, 1925.

231. Bloch, B.: Krankendemonstrationen aus der Dermatologischen Klinik Zürich, Schweiz med. Wchnschr., 56:404-405, 1926.

232. Bloch, Bruno, and Stauffer, H.: Skin diseases of endocrine origin [dyshormonal dermatoses]: poikilodermalike changes in connection with underdevelopment of the sexual glands and dystrophia adiposogenitalis, Arch. Dermat. & Syph., 19:22-34, 1929.

233. van Bogaert, M. Ludo: La mélanose neurocutanee diffuse hérédofamiliale, Bull. Acad. Roy. de med. de Belgique, 13:397-428, 1948.

234. Carney, R. G.: Incontinentia pigmenti; report of 5 cases and review of the literature, A.M.A. Arch. Dermat. & Syph., 64:126-135 (Aug.) 1951.

235. Cockayne, E. A.: Inherited Abnormalities of the Skin and Its Appendages, London, Oxford University Press, 1933.

236. Cole, H. N.; Giffen, Horace K.; Simmons, J. T., and Stroud, George M., III.: Congenital cataracts in sisters with congenital ectodermal dysplasia, J.A.M.A., 129:723-728 (Nov. 10) 1945.

237. Curth, Helen Ollendorff: Follicular atrophoderma and pseudopelade associated with chondrodystrophia calcificans congenita, J. Invest. Dermat., 13:233-247, 1949.

238. Duke University Case, Baltimore, Washington Dermat. Soc., 13:XI, 1948.

239. Findlay, G. H.: On the pathogenesis of incontinentia pigmenti, with observations on an associated eye disturbance resembling retrolental fibroplasia, Brit. J. Dermat., 64:141-146, 1952.

240. Franceschetti, A.: Les dysplasies ectodermiques et les syndromes héréditaires apparentés, Dermatologica, 106:129-156, 1953.

241. Franceschetti, A., and Jadassohn, W.: A propos de l'incontinentia pigmenti, délimination de deux syndromes différents figurant sous le mêm terme, Dermatologica, 108:1-28, 1954.

242. Franklin, A. White: Incontinentia pigmenti, Proc. Soc. Med., 45:219, 1952.

243. Gasteiger, H.: Über augenveränderungen bei Melanoblastosis Bloch-Sulzberger [Ocular changes in Bloch-Sulzberger Melanoblastosis], Deutsche ophth. ges., 57:292-294, 1951.

244. Gerard, R.: Les manifestations oculaires de l'incontinentia pigmenti (Ocular changes in incontinentia pigmenti), Bull. Soc. belge d'opht., 89:423-425, 1948.

245. Gottron, H.: Ichthyosis foetalis larvata, Zentralbl. Haut. -und Ges. Krankheiten, 43:612, 1933.

246. de Graciansky, P., and Boulle, S.: Atlas de Dermatologie, Paris, Maloine, 1952.

247. Guillaumat, L., and Maeder, G.: Syndrome de Rothmund (présentation de malade), Arch. opht., 3:353, 1949.

248. Haber, H.: Incontinentia pigmenti (Bloch-Sulzberger): A case history. Brit. J. Dermat., 61:104-105, 1949.

249. Habermann, P., and Fleck, M.: Über das Rothmund-Syndrom, Ztschr. Kinderh., 77:306-321, 1955.

250. Haxthausen, H.: Re-demonstration of a 1-year-old child with bullous, lichenoid eruption, Acta Dermato-Vener., 25:528, 1945.

251. Heilesen, Bjorn: Incontinentia pigmenti (two new cases), Acta Derma-Vener., 28:544-552, 1948.

252. Higuchi, K.; Urabe, H.; Kironaka, T., and Hirata, T.: Cited by Kitamura et al., 1954[259].

253. Höra, J.: Nebennierenmarkaplasie und eigenartige Hautpigmentanomalie, Zentralbl. allg. Path., 81:52-58, 1943.

254. Ito, Minor: Studies on melanin. X. Incontinentia pigmenti (Bloch-Sulzberger), J. Exper. Med., 54:67-72, 1951.

255. Jackli, W.: Ein Fall von infantiler Poikilodermie (Atrophodermia reticularis cum incontinentia pigmenti) kombiniert mit Alopecie, Mikrodontie und frühzeitiger Cataracta complicata, Monatschr. Kinderh., 78:73-81, 1939.

256. Jaramillo, Roberto; Manterola, Alejandro, and Rosselot, Jorge: Incontinentia pigmenti, Rev. chilena de pediat., 19:654-662, 1948.

257. Kawamura: Cited by Kitamura et al., 1954.[259]

258. Kitamura, K., and Fukushiro, R.: Jap. J. Dermat., 61:119, 1951.

259. Kitamura, Kanehiko; Fukushiro, Ryoichi, and Miyabayashi, Tetsu: Incontinentia pigmenti in Japan: introduction of 21 observed cases, A.M.A. Arch. Dermat. & Syph., 69:667-673, 1954.

260. Kuhling, F.: Incontinentia pigmenti, Wurzburg, Thèse, 1949.

261. Lapiere, S.: Incontinentia pigmenti, Arch. belges Dermat. & Syph., 7:156-517, 1951.

262. Lechleuthner, H.: Ueber eine seltene systematische Dermatose, München, Thèse, 1925.

263. Lutz, W.: Erythrodermia leukämica, Schweiz. med. Wchnschr., 58:1118, 1928.

264. Maeder, Guy: Le syndrome de Rothmund et le syndrome de Werner, Ann. ocul., 182:809-854 (Nov.) 1949.

265. Miescher, G.: I. Hauptthema—Theme principal der heutige Stand der peroralen Chemotherapie der Gonorrhoe, Dermatologica, 83:1-19, 1941.

266. Moncorps: Krankenvorstellungen. Incontinentia pigmenti bei einem 2¾ jährigen Knaben, Zentralbl. Haut- Ges. Krankheiten, 54:291, 1936.

267. Pierini, Luis E.; Garcia, Lucio A.; Pomposiello, Ismael, and Rey, Osvaldo: Incontinentia pigmenti (Bloch-Sulzberger) Rev. Argent. Dermatosif., 29:181-190 (Sept.) 1945.

268. Rothmund, August: Ueber Cataracten in Verbindung mit einer eigentümlichen Hautdegeneration, Arch. Ophth., 14:159-182, 1868.

269. Schäfer, Erich: Zur Lehre von den congenitalen Dyskeratosen, Arch. Dermat. u. Syph., 148:425-432, 1925.

270. Schynder, Walter F.: Ueber Kataract im kindesalter bei glerchzeittigem Vorkommen von Poikilodermia atrophicans. Familiares Vorkommen in der Schweiz (Rothmund'sche Krankheit), Schweiz. med. Wchnschr., 65:719-721, 1935.

271. Scott, J. Graham; Friedmann, A. I.; Chitters, M., and Pepler, W. J.: Ocular changes in the Bloch-Sulzberger syndrome (incontinentia pigmenti), Brit. J. Ophth., 39:276-282, 1955.

272. Seefelder, R.: Über familiäres Auftreten von Katarakt und Poikilodermie, (Poikilodermia vascularis atrophicans Jakobi), Ztschr. Augenh., 86:81-97 (May) 1935.

273. Seidlmayer, Hubert: Über ein neues Syndrom multipler Abartung: Pigmentanomalie, kongenitale ektodermale Dysplasie, Nebennierenmarkaplasie, Ztschr. Kinderh., 63:451-487, 1942.

274. Siegrist, August: Der graue Altersstar, seine Ursachen und seine nichtoperative Behandlung, Berlin, Urban & Schwarzenberg, 1928.

275. Siemens, Hermann Werner: Die Melanosis corii degenerativa, eine neue Pigmentdermatose, Arch. Dermat. u. Syph., 157:382-391, 1929.

276. ———: Studien über Vererbung von Hautkrankheiten. XI. Ichthyosis congenita, Arch. Dermat. u. Syph., 158:111-127, 1929.

277. ———: Die Vererbung in der Ätiologie der Hautkrankheiten, In Josef Jadassohn's Handbuch der Haut- und Geschlechtskrankheiten, Berlin, J. Springer, 1929, vol. III.

278. Sobel, Nathan: Incontinentia pigmenti (report of case), Arch. Dermat. & Syph., 58:467-468, 1948.

279. Strebel, J.: Ueber zweiseltene Hornhauterkrankungen, Beitr. Augenh., 84: 351-404, 1913.

280. Sulzberger, Marion B.: Über eine bisher nicht beschreibene congenitale Pigmentanomalie. (Incontinentia pigmenti), Arch. Dermat. u. Syph., 154:19-32, 1928.

281. ———: Incontinentia pigmenti (Bloch-Sulzberger). Report of an additional case, with comment on possible relation to a new syndrome of familial and congenital anomalies, Arch. Dermat. & Syph., 38:57-69, 1938.

282. Thomson, M. Sidney: Poikiloderma congenitale, Brit. J. Dermat., 48:221-234 (May) 1936.

283. Uebel, H.; Ludwig, A., and Korting, G. W.: Zur Kenntnis der Incontinentia pigmenti Bloch-Sulzberger, Arch. Dermat. u. Syph., 190:114-124, 1950.

284. Watanabe: Cited by Kitamura et al., 1954.[259]

285. Werner, C. W. O.: Ueber Katarakt in Verbindung mit Sklerodermie, Kiel, Thèse, 1904.

286. Zinsser: Iconogr. Dermat., 5:219, 1910.

RETINAL DETACHMENT SURGERY: SCLERAL SHORTENING BY OUTFOLDING WITH TITANIUM CLIPS

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FOR some time prior to 1953 I performed scleral shortening surgical procedures in complicated cases of retinal detachment that had failed to respond well to classical diathermy. I used both lamellar and full-thickness resections of the sclera in a sufficient number of cases to convince myself that these techniques were unnecessarily complicated and lengthy, and it occurred to me that it might be possible to shorten the sclera just as effectively by more simple procedures. In addition, I found the full-thickness resection of the sclera excessively traumatic to the eye, not infrequently causing serious complications.

In 1953 and 1954 I used a new technique for scleral shortening in eleven eyes. It consisted in folding the sclera outward and maintaining the fold by means of mattress sutures. The sclera near the fold was treated with electrodiathermy coagulations. An operation very similar to the one I used on humans, but had not reported, was described by Dr. William G. Everett[2] in the June 1955 issue of the *A.M.A. Archives of Ophthalmology* in a paper entitled "A Preliminary Report on a Scleral Shortening Operation." His findings were based on experimental and clinical studies. At the end of his paper Dr. Everett mentioned that since its preparation, he had learned that I had coincidentally developed a similar operation. As stated by Dr. Everett, I had used this type of scleral outfolding in eleven patients and found it unsatisfactory because the needles and suture material, which had to pass through the scleral fold, occasionally injured the choroidal vessels with subsequent severe intraocular hemorrhages. I also found that after some time the scleral sutures became loose and the fold disappeared, with a tendency to recurrence of the detachment. This flattening of the scleral fold after several months was verified clinically in cases in which the detachment recurred and another operation was performed.

In order to overcome these objections to the use of sutures to maintain the scleral fold, I developed a new method. The fold is maintained with nonrust metal clips which do not penetrate the sclera, thus avoiding injury to the intraocular blood vessels. When the clips are adequately applied, the scleral fold is maintained indefinitely, as proved clinically in cases of surgically treated recurrent detachment.

This procedure for scleral outfolding, maintaining the fold with nonrust metal clips rather than silk or catgut sutures, was reported as a new instrument[1] at the Sixtieth Annual Session of the Ameri-

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Presented at the Sixty-Fourth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 11-16, 1959, Chicago.

can Academy of Ophthalmology and Otolaryngology held in Chicago, October 9 to 14, 1955. The report, "New Clips and Clip-Applying Forceps for Scleral Shortening Procedure," described a simple method of scleral shortening for the treatment of retinal detachment. Monel metal clips, 4 mm. long, 1 mm. wide, and with teeth measuring 0.25 mm., were used to hold the scleral fold. In this procedure the clips are applied after the subretinal fluid has been drained through a diathermocoagulation puncture made approximately at the equator and enlarged with a punctum dilator. The sclera near the fold is treated with diathermocoagulations and air is injected into the vitreous if the retina is still detached at the completion of the operation.

I used this method in five almost hopeless cases in which one or more operations for retinal detachment had failed. The results seemed to justify reporting it to the Academy: a simple technique that could be used by any ophthalmic surgeon in selected cases of retinal detachment in preference to other more complicated procedures for scleral shortening, with a prognosis as good or better than that offered by the other methods. Shortly thereafter, in two of the reported cases I observed persistent conjunctivitis with moderate chemosis and a mucopurulent discharge that did not respond to treatment and seemed to be caused by intolerance of the orbital tissues to the metal clips. This observation formed an important addendum when the report was published, with the advice that until the innocuity of the clips had been demonstrated by further work the procedure should be considered as still in the experimental stage.

Further study demonstrated titanium to be very well tolerated by the ocular and orbital tissues, causing even less reaction than silk, catgut, linen or other materials. Titanium clips have now been used in fifty-one cases of retinal detachment, and the results seem to compare favorably with those of other scleral shortening techniques. Although this limited number of cases does not give sufficient data to form a final opinion about the procedure, encouraged by the results already obtained, I am reporting it at this time to acquaint other ophthalmologists with the possibilities of this new approach to the treatment of retinal detachment, urging them to use it in selected cases to arrive at an unbiased evaluation of its merits.

TECHNIQUE

The retinal detachment should be carefully plotted and all retinal tears localized before the operation in order to place the scleral fold in the best possible location to seal the retinal rents.

The essential instruments for the operation are (1) titanium clips, 4 mm. long, 1 mm. wide, and with one 0.25 mm. tooth at each end, which overlap when the clip is closed (fig. 1, C and H); (2) a clip-applying forceps with grooved jaws to engage the clips (fig. 1, A and B), and a coil spring to provide enough tension to keep the jaws sufficiently taut to hold the clips snugly; (3) a double-ended punctum dilator with one sharp end and the other blunt, and (4) a 2-cc. syringe with a sharp 30-gauge needle to inject air or vitreous into the vitreous cavity when necessary at the end of the operation.

The operation may be performed under general or local anesthesia. If local anesthesia is used, akinesia of the orbicularis is obtained by injecting several centimeters of a 1 per cent solution of Xylocaine, following a modified Van Lint technique. The eye is anesthetised with a retrobulbar injection of about 3 cc. of Xylocaine, 2 per cent solu-

tion, with one drop of Adrenalin, 1:1000 per cubic centimeter, and a subconjunctival injection of a 2 per cent solution of Xylocaine with Adrenalin, 1:1000, in the area of the rectus muscles to be involved in the operation. Five units of hyaluronidase per cubic centimeter may be added to the anesthetic. If the palpebral fissure is small, a canthotomy will facilitate the operation, providing a larger operative field. An incision is made through the conjunctiva and Tenon's capsule about 7 to 8 mm. from the limbus, and these membranes are freed until a good exposure of the rectus muscles and sclera is obtained. A good view of the operative field is required. Therefore, if more than one quadrant of the sclera is involved in the operation, the muscle or muscles over this area should be disinserted. One of the muscles is disinserted and a traction 4-0 silk suture is placed on the scleral stump of the muscle for easy handling of the eye (fig. 2, A). For better manipulation of the eye, other 4-0 silk traction sutures may be placed under the medial and lateral rectus muscles if desired. The fold is placed more anteriorly or posteriorly, depending upon the location of the retinal tears. Generally it is made equatorially, posterior to the rent or rents, unless these are located beyond the equator toward the optic nerve, in which case it should be placed around the equator. The rents should be sealed later with meridional folds and diathermocoagulations extending toward the disc.

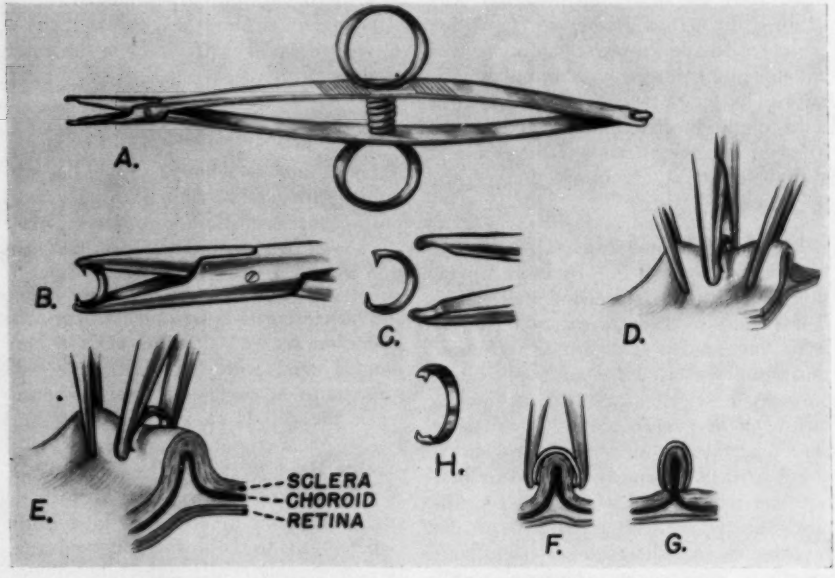

FIG. 1—(A) Clip-applying forceps. (B) Detail of the jaws holding the clip. (C and H) Titanium metal clip. (D, E, F, G) Application of the clips through the folded sclera.

It is essential in this procedure that the subretinal fluid be well evacuated to make the eye soft and permit formation

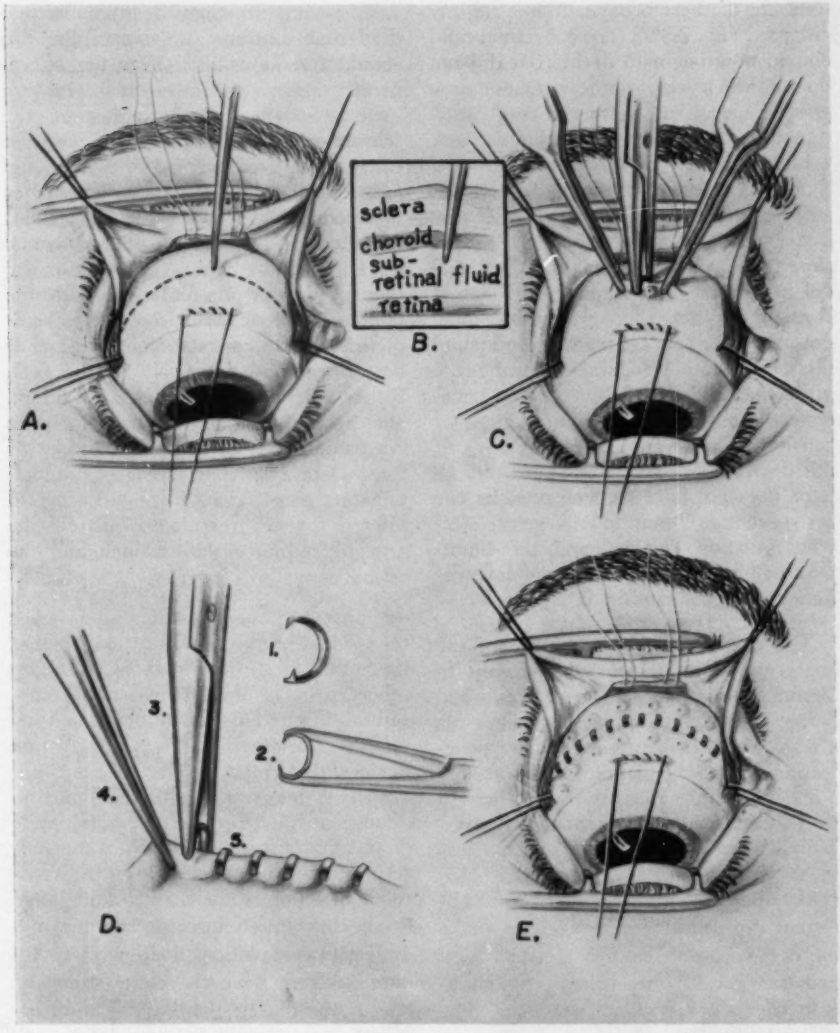

FIG. 2—Different steps of the operation of outfolding of the sclera with clips.

of the scleral fold. Contrary to the opinion expressed by most authors, I find the punctum dilator, when properly used, to be the best instrument to use in evacuating the subretinal fluid. A diathermocoagulation is made with a 1 mm. electrode in the sclera along the equator in the area corresponding to the greatest retinal elevation. With a sharp punctum dilator, which penetrates the eye deep enough to pass through the sclera and choroid, but not more than 1.5

mm., the diathermocoagulation is slightly enlarged (fig. 2, A). The subretinal fluid should now begin to drain if it did not do so when the diathermocoagulation was made. The diathermocoagulation puncture is then further enlarged with a blunt punctum dilator until subretinal fluid has been drained in an amount sufficient to permit the scleral outfolding. If no hemorrhage occurred at the time of the electrocoagulation, the careful use of the sharp and blunt dilators through the electrocoagulation should not cause bleeding. If the sharp punctum dilator is not introduced into the eye more than the 1.5 mm. needed to penetrate the choroid, there is no danger of causing retinal trauma, even in cases of flat detachment. The blunt punctum dilator does not have to penetrate the eye for more than 2 mm. to enlarge the electrocoagulation further, and the bluntness of the instrument should not produce retinal injury (fig. 2, B).

The evacuation of all or most of the subretinal fluid, which is important to obtain a good result, can be accomplished better by using a punctum dilator as already described, rather than by penetrating electrocoagulations which are often self-sealing, unless the sclera is very thin. This method of evacuating the subretinal fluid is more simple and less likely than a sclerotomy to cause choroidal hemorrhage. When sufficient subretinal fluid has been drained to make the eye soft, the sclera is grasped with two fixation forceps sturdy enough to hold the sclera without wobbling. The clip is held by the clip-applying forceps and is brought into position between the two fixation forceps holding the scleral fold (fig. 1, D and E; fig. 2, C). By closing the clip-applying forceps, the clip is left securely in place over the outfolded sclera (fig. 1, F and G). For the fold to be fully effective, the clip should be well placed tightly against the fold.

To permit drainage of any subretinal fluid that continues to escape, the clip should not be applied over the scleral perforation, as the intraocular pressure increases with the shortening of the sclera through the formation of the fold. Clips are applied every 2 to 3 mm. (fig. 2, D) until a fold of desired length has been obtained or until the intraocular pressure has attained a fairly normal level. The last clip is applied to seal the scleral opening enlarged with the punctum dilator. After the fold is completed, electrocoagulations are made posteriorly and anteriorly to the fold. Either a 0.5 mm. electrode or a 1.0 mm. one, applied obliquely, is used in order not to penetrate the choroid in cases of thin sclera (fig. 2, E). Electrocoagulations are placed at both ends of the fold toward the ora serrata to encircle the area of retinal elevation, including the retinal rent.

Ophthalmoscopic examination when the fold is completed will reveal whether the retina is still elevated. If it is, another effort is made to drain the subretinal fluid. This is accomplished by means of another diathermocoagulation made with a 1.0 mm. electrode and enlarged, if necessary, with the punctum dilator, as described before. If, in spite of this further attempt to drain the subretinal fluid, the eye remains soft, a vitreous injection is made using a 30-gauge needle introduced into the eye through an electrocoagulation done with a 1.0 mm. electrode near the scleral stump of one of the muscles (fig. 3). The effectivity of the vitreous does not seem to be altered by injecting it into the eye through a 30-gauge needle, and this method is more simple than employing thicker needles through sclerotomies, for they are more traumatic and it may be necessary to close the puncture points with sutures. When the retina is still elevated at the completion of the opera-

tion but the intraocular pressure is near normal, vitreous injection may cause secondary glaucoma. In these cases it is preferable to inject, instead, a small amount of air into the vitreous through a 30-gauge needle in a manner similar to that described before for the vitreous injection, in order to push the elevated retina against the choroid. The operation is finished by suturing the muscle or muscles to their scleral stumps with 4-0 mild chromic catgut and closing the conjunctival and Tenon's capsule incision with 6-0 mild chromic catgut.

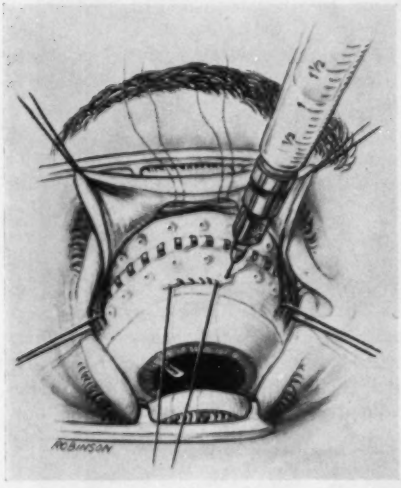

FIG. 3—Injection of air or vitreous into the eye.

A variation of this technique using meridional folds, as illustrated in figure 4, is indicated particularly in cases of bullous detachment with an extensive rent involving a large portion of one quadrant and with no tendency of the retina to reattach because of permanent retinal folds, retinal retraction or pulling vitreous bands. It is also indicated when the retinal tear is located posteriorly to the equator and an equatorially placed

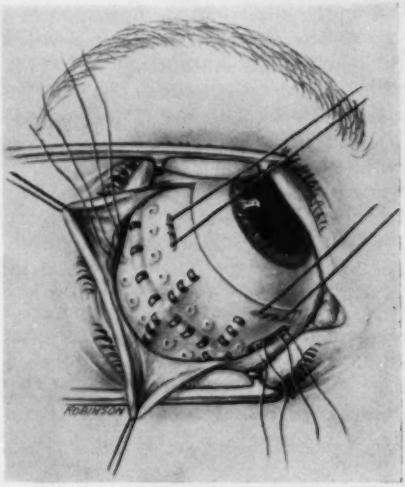

FIG. 4—Variation of the technique of scleral outfolding, showing equatorial and meridional folds.

scleral fold is not likely to seal the rent. In these cases the sclera may be pushed in by one, two or more meridional folds. This technique may also be used in cases of reoperation for recurrent retinal detachment if the retina shows characteristics similar to those outlined above.

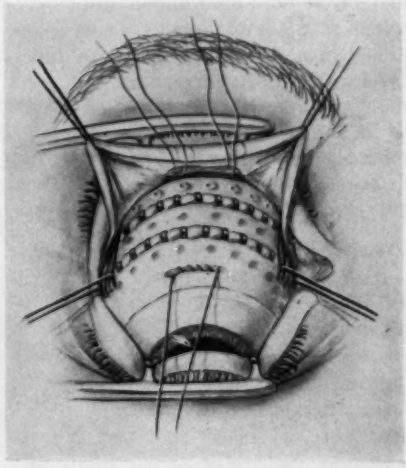

FIG. 5—Shows two scleral folds made in two different operations.

Figure 5 illustrates the formation of a second scleral fold in an eye in which a previous scleral outfolding with clips failed because the retinal tear was located posteriorly to the fold. A second scleral fold placed more posteriorly was required to seal the rent.

EVALUATION OF RESULTS

As mentioned at the beginning of this paper, the first five cases operated upon showed intolerance to the Monel metal clips. In two cases several of the clips had to be removed, and in one case all of the clips had to be taken off before the eyes became quiet. In spite of this foreign body reaction, the retina remained attached in three of these five cases, which were selected because of an almost hopeless prognosis, to test the tolerance of the ocular tissues to the metal clips. These encouraging results justified further investigation of this procedure in a larger group of patients in order to evaluate its merits, selecting, however, a metal for the clips that would be tolerated by the ocular tissues. This proved to be titanium, and clips of this metal have been used to date in 61 operations in 51 eyes. Some eyes were operated upon twice and one, three times. Over a period of three and a half years the eyes and orbital tissues have tolerated the titanium clips as well as they have other foreign materials used in retinal detachment surgery, including silk, catgut, and linen.

For over two years, in thirty selected cases of retinal detachment with a not too favorable prognosis in which classical diathermy was not likely to succeed and scleral shortening procedures seemed indicated, the titanium clips were used mainly to test, in a sufficient number of eyes over a period of time, the reaction of the ocular tissues to the implantation of this foreign material. When it was verified in a period of over two years that the titanium clips were well tolerated and the results obtained were encouraging, 21 more scleral outfolding operations were performed to evaluate the merits of the procedure in other cases with a more favorable prognosis but again in which scleral shortening rather than classical diathermy was indicated. The scleral outfolding with clips has now been used in 67 operations in a total of 56 eyes. Monel metal clips were used six times in the first five eyes, and titanium clips 61 times in the remaining 51. Nine eyes required two outfolding operations, and one required three. Of the 56 cases observed over a period from three months to four years, the retina remained detached after one or more scleral outfolding operations in 26 cases. In two of these 26 cases recorded as failures, including those in which Monel metal clips were used, a second operation was advised but the patients went elsewhere for further treatment. Two operations were required in three of the cases that failed to respond to this surgical technique, and in one case three operations were performed unsuccessfully. In thirty cases (53.6 per cent) the retina remained attached. Six of the thirty successful cases required two scleral outfoldings with clips to effect a retinal reattachment.

Of the 26 cases in which the operation failed, one or more classical diathermy operations had been performed in nine, and seven of these 26 eyes were aphakic. Of the 30 cases in which the retina remained attached, eight had had previous classical diathermy operations for retinal detachment, and six eyes were aphakic. It is interesting to note that of the first 35 cases selected for this operation because of a not too favorable prognosis, 22 (62.8 per cent) were failures. Of the remaining 21 cases with better prognosis, only four (19.04 per cent)

were failures, the percentage of successes increasing in direct proportion to improvement in the prognosis. In addition to what may have been insufficient experience in the use of this new method, a predisposing factor for the failures in the first 35 cases could have been the application of the clips at too great intervals of 6 to 7 mm., which diminished the effectivity of the fold. However, I believe that by far the principal cause of failure in these cases was the poor prognosis. In the later group of cases, in which the eyes were more favorable for surgery, the clips were placed much closer together at 2 to 3 mm. and the scleral fold was more effectively maintained.

Figures 6 to 13 reproduce roentgenograms of orbits, showing the clips used for scleral outfolding operations. Figure 6 shows one of the earliest cases, with the clips placed about 7 mm. apart.

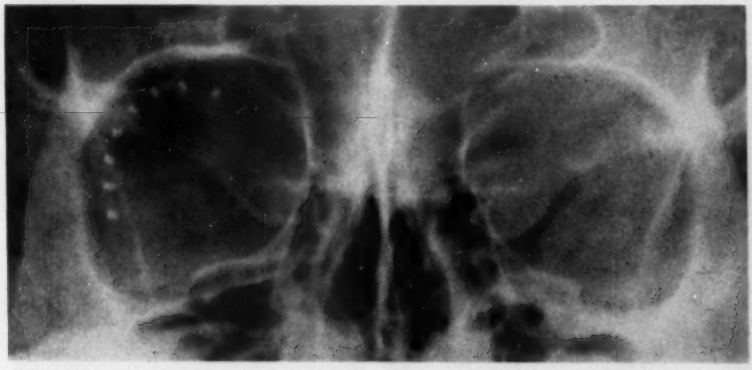

FIG. 6—Roentgenogram showing one of the earliest cases of scleral outfolding with clips placed about 7 mm. apart.

FIG. 7—Roentgenogram showing a case of scleral outfolding operated at a later date with the clips placed closer together.

FIG. 8—Roentgenogram showing four clips in a short fold.

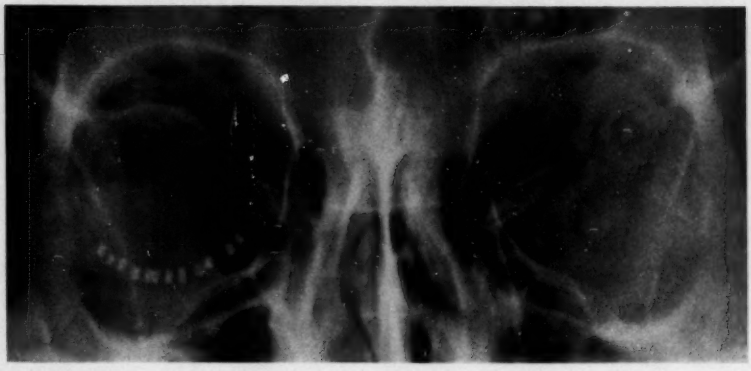

FIG. 9—Roentgenogram of clips in a fold required for the treatment of slightly over one quadrant.

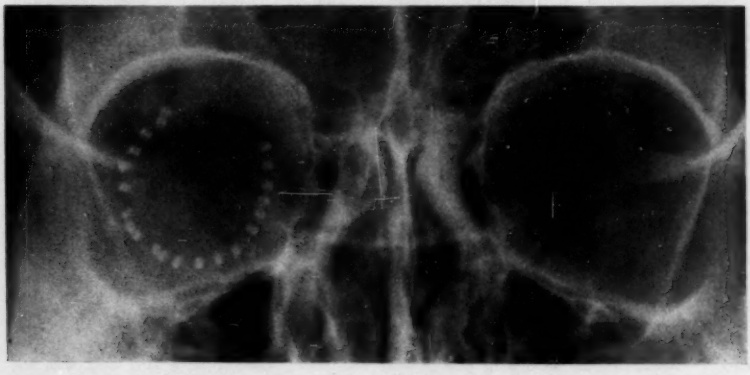

FIG. 10—Roentgenogram showing clips in a fold extending over three quadrants.

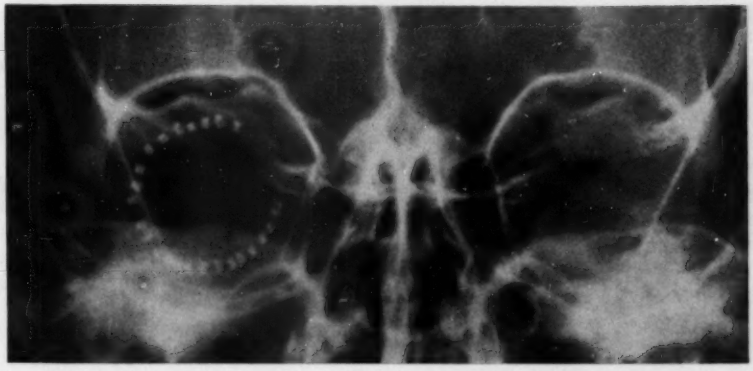

FIG. 11—Roentgenogram showing clips in a fold extending over three quadrants.

FIG. 12—Roentgenogram showing clips placed in scleral folds equatorially and meridionally.

FIG. 13—Roentgenogram showing clips used in two equatorial folds and one meridional fold made in two separate operations.

Figure 7 illustrates a case operated upon at a later date and shows how much closer together the clips were applied. The roentgenograms also show how in some cases only a few clips were necessary to treat one quadrant (fig. 8), while in other cases (figs. 9, 10 and 11) a longer fold, involving two or even three quadrants, was required. Figure 12 shows clips placed equatorially and meridionally in scleral folds. Figure 13 shows clips used in two equatorial and one meridional fold made in two different operations.

The visual results obtained with this operation seem to parallel those obtained with other successful operations for retinal detachment. In cases in which the macula had not been seriously affected, final vision was good. Of course, when the macula was seriously involved with degenerative changes, vision remained poor in spite of the successful reattachment of the retina.

No intolerance was demonstrated in eyes in which titanium clips were used, and none of these clips had to be removed. No tendency of the clips to perforate the sclera or to migrate within the eye was noted in any of the cases in which either Monel metal or titanium clips were used.

The effectiveness of the operation, as with any other scleral shortening procedure, seems to be caused by the decrease in the surface area of the sclera and in the reduction of the volume of the eye. Ophthalmoscopic examination, both during the operation and postoperatively, has demonstrated that the retina is not included in the fold. On the other hand, it is possible that the choroid may be enclosed in the fold, at least in some places, although I have not been able to verify this possibility either experimentally or by microscopic examination of eyes treated by this method. However, if the results obtained are satisfactory, it seems to be of little consequence whether or not the choroid is included in the fold.

COMPLICATIONS

Two patients developed increased ocular tension that responded well to miotics. It was not possible to determine whether this increase in tension was the result of too great a reduction in the size of the eye, or whether it was caused by the vitreous injected into the two eyes in which this complication occurred. In several cases, I used lyophilized vitreous supplied by Dr. John Harry King, Jr.[3] The eyes in which this vitreous was used seemed to follow the same course as those treated with refrigerated vitreous from cadaver eyes. Other complications included one case of severe vitreous hemorrhage and several cases of persistent vitreous floaters. Not a single eye was lost because of orbital infection or endophthalmitis. No intolerance to the foreign material was noted in any of the eyes in which titanium clips were used, and none of these clips had to be removed.

CONCLUSIONS

From this limited series of cases in which scleral outfolding with titanium clips was used for the treatment of retinal detachment, the following conclusions may be drawn:

1. The titanium clips used to hold the sclera do not cause foreign body reaction in the eye or orbital tissues. They seem to be as well tolerated as silk, catgut, linen, and other materials.

2. Scleral outfolding with clips is a more simple and a shorter procedure

than scleral outfolding with sutures. It does not incite hemorrhage and maintains the fold better than sutures.

3. The operation is also more simple and has proved to be more effective than either lamellar or full-thickness resections, weakening the scleral wall less than either of these or scleral buckling.

4. The simplicity of the operation permits a greater number of ophthalmic surgeons to perform it without the highly specialized training and skill required for some of the newer operations devised in this field of ocular surgery.

COMMENTS

I consider this report to be a preliminary one to follow the new instrument presentation before the American Academy of Ophthalmology and Otolaryngology in Chicago in 1955. I would not have presented it at this time if I had not been forced to do so by certain conflicting references in the literature to this technique, apparently made with no understanding of its mechanics and possibilities when applied in selected cases in which classical diathermy is apt to result in failure. Further study of this technique may result in improvements and in establishing definite indications for its use as well as definite contraindications.

It is not the purpose of this paper to give a detailed case history of each patient. This will be done at a later date when more complete study, comparison with other methods, particularly the new buckling procedures, and more cases have been under observation for a longer period of time to determine whether presence of the metal clips in the eye is as innocuous as it seems to be at the date of this report.

I realize that the scleral outfolding operation with titanium clips has not been sufficiently tested to draw final conclusions regarding its merits. I am convinced, however, that scleral outfolding with clips represents an improvement over other methods of scleral shortening, including lamellar and full-thickness resections of the sclera and outfolding with sutures. However, until other ophthalmic surgeons have had an opportunity to evaluate this method without the unavoidable prejudice of the author, it will not be possible to state definitely whether it represents a step forward in retinal detachment surgery or is just another attempt, in this era of nonconformism, to increase the percentage of successful outcomes, especially in those types of cases that do not respond well to previously known surgical procedures.

SUMMARY

This is a follow-up of the report on a new instrument presented before the Academy in Chicago in 1955,[1] with a brief summary of the results obtained in 56 selected cases of retinal detachment. The main purpose of this follow-up report is to offer the experience of the author to other ophthalmic surgeons in the hope that they will contribute with their own studies to make possible a full and unbiased evaluation of this procedure.

The clips and clip-applying forceps are manufactured by Storz Instrument Company, 4570 Audubon Avenue, St. Louis 10, Missouri.

REFERENCES

1. Castroviejo, Ramon: New clips and clip-applying forceps for scleral shortening procedure, Tr. Am. Acad. Ophth. & Otol., 60:483-485 (May-June) 1956.

2. Everett, William G.: A new scleral shortening operation; preliminary report, A.M.A. Arch. Ophth., 53:865-869 (June) 1955.

3. King, John Harry, Jr.: Keratoplasty: experimental studies with corneas preserved by dehydration, Tr. Am. Ophth. Soc., 54: 567-609, 1956.

DISCUSSION

WILLIAM G. EVERETT, M.D., Pittsburgh: I appreciate this opportunity to discuss Dr. Castroviejo's paper. My interest in this subject, as you know, goes back to the original article in 1955, in the Archives of Ophthalmology, and the presentation before this group in 1956 of a motion picture of this procedure utilizing silk sutures.

Dr. Castroviejo has modified his feelings about this operation over the years and has changed his technique. I, too, have developed new thoughts on the procedure after having operated in many clinical cases using this technique.

One point I should like to make clear is with regard to whether or not the choroid is incarcerated within the outfold. Dr. Castroviejo's paper states that it is not known experimentally or clinically whether this is so, and his diagram shows the choroid clearly incarcerated within the wound.

[Slide] My first slide shows here an immediate postoperative picture of an outfolding in a dog. The vitreous is a normal vitreous, creating a little more pressure than encountered in a clinical detachment, but you will notice that in the outfold procedure the choroid is not present.

[Slide] In the next slide a picture of a rabbit's sclera clearly shows that the vascular structure of the choroid does not become involved in the outfold if it is properly treated.

[Slide] The next slide shows that the pigment cells of the suprachoroidal area do migrate within the base of the fold about four days postoperatively, but the vascular structure is undisturbed.

Dr. Castroviejo has given up the sutures because of intraocular hemorrhages due to pierced choroid. [Slide] I have been very careful to grasp the episclera after decompression, then to take a very small bite with a Castroviejo-type forceps [slide]; and then, by increasingly larger bites, the sclera is brought into an outfold position, at the same time milking the choroid from this fold. In numerous clinical cases I have excised the top of my fold above the sutures and have not encountered an incarcerated choroid. I feel this is important because once the fold is created, one could very simply use the titanium clip as described by Dr. Castroviejo. However, in an area of thin sclera, as in an area of staphyloma, one might even perforate a scleral vessel with a 0.25 mm. clip and cause serious intraocular hemorrhage. I believe that if this maneuver of gradually outfolding the sclera, simple as it is, is observed, choroidal hemorrage need not occur.

[Slide] In applying a scleral suture, one depresses the sclera in front of the point of expected emergence with a muscle hook which depresses and pushes the choroid back and away from the sutures. All of my operations have been done with suturing, and I have had no experience with the metallic clips.

[Slide] This slide demonstrates one reason why I have altered my thoughts on outfolding. Here is the retina adjacent to a scleral outfold in a donor eye. You will note that the retina is thrown into irregular rolls and folds, and this is typical of a scleral outfold procedure which reduces volume in excess of surface area. If you are dealing with a detachment in which the retina is of normal surface area and you markedly reduce the volume, there is a tendency for the retina to be thrown into an irregular fold. If your diathermy is properly applied on either side, this may be of no consequence, but if you have a large flap and the flap tends to be within one of these folds, then you may be headed for trouble. I feel that one cannot use this procedure for all detachments that require shortening. There are those in which one does not want to sacrifice surface area as opposed to volume.

[Slide] This slide shows a fundus with a silicon tube used as a scleral implant. When we are desirous of utilizing the full surface area of the detached retina, we can do this and still markedly reduce volume without sacrificing surface area. You will note that the tube applied in this manner allows the retina to lay over it in one sweeping smooth fold.

[Slide] My second opinion about the scleral outfold is one with which you may not agree, but I have been impressed by the fickleness of penetrating diathermy. I operated on this patient some time ago, and I think you will agree that this is reasonably satisfactory diathermy, but even with this there is a sharp contrast going from the diathermized area to the adjacent retinal area. There is a tendency for retinal breaks to occur if the diathermy is not uniform or is excessive.

[Slide] The currents that are required to provide a uniform treatment are unpredictable when you are using penetrating diathermy.

You might be interested to note in this slide that the pigmented stria underlies the retinal roll. The retinal rolls settle out and leave behind a pigmented stria such as this is. So far as this area is concerned, the case was a success. A second operation was necessary in an entirely different area.

[Slide] Here is a similar example of the great variability in diathermy when it is applied in a partially penetrating or a penetrating manner.

[Slide] This is a slide showing a lamellar undermine beneath a tear and choroidal diathermy. You will note the uniform distribution of pigment in healing. When you apply very light diathermy on the suprachoroidal area, you get a uniform area of treatment. This hole is still wide open, but it is well maintained. Underneath the hole a silicon plate facilitates the contact of the choroid with the retina.

I feel that there is a place for scleral outfolding, but I agree with Dr. Castroviejo when he says "in selected cases," and to me this is not a choice between classic diathermy and scleral shortening but rather a choice among those cases that require scleral shortening. Certainly if the clips prove to be completely innocous, this should prove to be an improvement on the basic procedure of scleral outfolding.

I would add one other point. I feel that time is on my side and time is of no importance when one attempts detachment surgery. I feel that if you wish to undertake the responsibility of detachment surgery, you must at the same time never hurry your procedure. If one has a new procedure which takes less time and is equally good, this is fine; but the ultimate goal is the preservation of vision and the salvation of those remaining patients with detached retinas for whom we have yet to find the answer.

PASSIVE IMMUNIZATION AGAINST PSEUDOMONAS INFECTION OF THE CORNEA

J. WALLACE MCMEEL, M.D.
RONALD M. WOOD, PH.D.
BALTIMORE, MARYLAND

BY INVITATION

PSEUDOMONAS infections of the human cornea are generally severe and may rapidly progress to endophthalmitis, resulting in permanent loss of vision within twenty-four to forty-eight hours. Antibiotic therapy has been disappointing in the control of this infection.

Having demonstrated that following active immunization with Pseudomonas aeruginosa,[1] rabbits were highly immune to corneal challenge with this organism, experiments were undertaken to evaluate the effectiveness of passive immunization prior to and several hours following corneal challenge.

MATERIALS AND METHODS

Pseudomonas-Immune Gamma Globulin

Twenty normal rabbits were immunized over a three-week period by giving three intravenous injections per week consisting of 0.1, 0.3 and 0.5 ml. of a heat-killed suspension of Pseudomonas aeruginosa standardized to a light transmittance of 50 per cent.[1] Seven days following the last immunizing injection, the animals were bled by cardiac puncture. The gamma globulin was precipitated from the serum by half saturation with ammonium sulfate and, after dialysis, adjusted to a protein concentration of 28 mg. per milliliter. This globulin had a titer of 1:320 for Pseudomonas aeruginosa by the tube agglutination technique. The gamma globulin solution was sterilized by Seitz filtration before use.

Technique of Corneal Challenge

The eyes were anesthetized with 1 per cent Pontocaine. Using a trephine with a diameter of 5 mm., an incision 0.2 mm. deep was made in the center of the cornea. Two drops of an actively growing 24-hour culture of Pseudomonas aeruginosa were then placed on the prepared cornea.

Experimental Groups and Dosage

All injections of the Pseudomonas-immune gamma globulin were given intramuscularly in the thigh muscle in a single dose. All rabbits were albinos, averaging five pounds in weight. Eight groups of rabbits were treated as outlined in table I. Groups 1 to 4 inclusive each received increasing amounts of Pseudomonas-immune gamma globulin forty-eight hours prior to corneal challenge. Groups 5 to 7 inclusive received 140 mg. of Pseudomonas-immune gamma globulin at intervals of six, twelve or twenty-four hours following corneal challenge. Group 8 served as controls.

From the Wilmer Ophthalmological Institute of the Johns Hopkins Hospital and University, Baltimore. This work was supported by a grant (B43-C11) from the Department of Health Education and Welfare, Public Health Service National Institute of Health, and in part by a grant from the Knights Templar Eye Foundation.

Presented at the Sixty-Fourth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 11-16, 1959, Chicago.

TABLE I

TREATMENT OF EXPERIMENTAL GROUPS WITH PSEUDOMONAS-IMMUNE GAMMA GLOBULIN

GROUP	NO. OF RABBITS	TIME OF TREATMENT RELATIVE TO CHALLENGE	INTRAMUSCULAR DOSE (MILLIGRAMS OF GLOBULIN PROTEIN)
1	4	48 hours before	14
2	4	48 hours before	28
3	4	48 hours before	70
4	8	48 hours before	140
5	4	6 hours after	140
6	4	12 hours after	140
7	4	24 hours after	140
8	4	Controls—untreated	None

RESULTS

The inflammatory reaction of the eyes of all the groups was essentially the same for the first twenty-four hours, although the animals given 70 mg. and 140 mg. of gamma globulin prior to challenge seemed to have slightly more inflammation at six hours post challenge. It was not until forty-eight hours following challenge that the difference became evident between the controls and those given a protective amount of Pseudomonas-immune gamma globulin prior to challenge.

There was a fairly definite break point in the protection afforded by the dose of gamma globulin given prior to challenge. Those animals given 14 mg. (group 1) or 28 mg. (group 2) had only partial protection, whereas those given either 70 mg. (group 3) or 140 mg. (group 4) had good protection in every case.

In the groups of animals given 140 mg. of gamma globulin six, twelve, or twenty-four hours after challenge, the alteration of the natural course of the infection was not evident until twenty-four to seventy-two hours after the gamma globulin had been given. Although there was not complete protection, over one half of the eyes had no residua at all. There was no significant difference in the final results observed in the group of animals given Pseudomonas-immune gamma globulin at either six, twelve or twenty-four hours following challenge (table II, figs. 1-3).

DISCUSSION

It is evident that the immune globulin protecting the corneas against infection arrived by an avascular route. The inflammation of the eye secondary to the challenge inoculation probably contributed to the entry of the immune gamma globulin into the cornea.

Since the progress of the infection was altered about forty-eight hours after the globulin was given, this apparent-

TABLE II

EFFECT OF INTRAMUSCULAR PSEUDOMONAS-IMMUNE GLOBULIN ON CORNEAL CHALLENGE WITH PSEUDOMONAS AERUGINOSA

GROUP NUMBER	TIME GIVEN	GAMMA GOBULIN (mg.)	NO. OF EYES	NO RESIDUA	NEBULA OR SMALL LEUKOMA	DENSE OPACITY AND VASCULARIZATION	PROTECTED EYES/ TOTAL EYES
1	48 hours before challenge	14	8	1	2	5	1/8
2	48 hours before challenge	28	8	2	3	2	2/8
3	48 hours before challenge	70	8	8	0	0	8/8
4	48 hours before challenge	140	16	16	0	0	16/16
5	6 hours after challenge	140	8	4	2	2	4/8
6	12 hours after challenge	140	8	5	1	2	5/8
7	24 hours after challenge	140	8	5	1	2	5/8
8	Controls		8	0	1	7	0/8

ly was the time required for intramuscularly administered antibody to reach the corneal stroma in effective concentration.

Gamma globulin given forty-eight hours prior to challenge was very effective in quantities of 70 mg. and 140 mg. The small prechallenge doses of globulin were less effective, giving only partial protection (table II).

When 140 mg. of Pseudomonas-immune globulin was given at six, twelve or twenty-four hours after challenge, fourteen out of twenty-four eyes were completely protected. A more potent immune globulin, either alone or in conjunction with suitable antibiotics, might reasonably be expected to give better results.

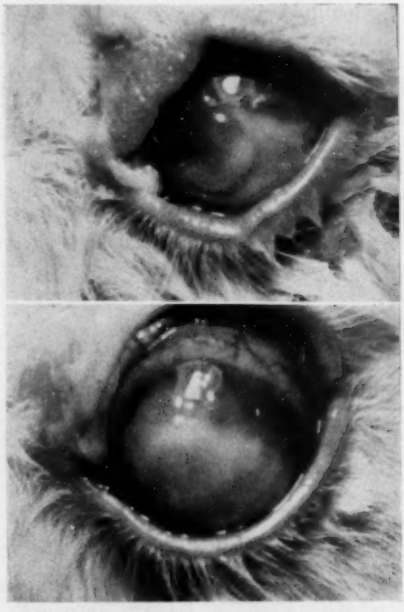

FIG. 1—Control group. Appearance seven days following challenge with Pseudomonas aeruginosa.

SUMMARY

Complete protection against Pseudomonas corneal infection was obtained when an adequate amount of Pseudo-

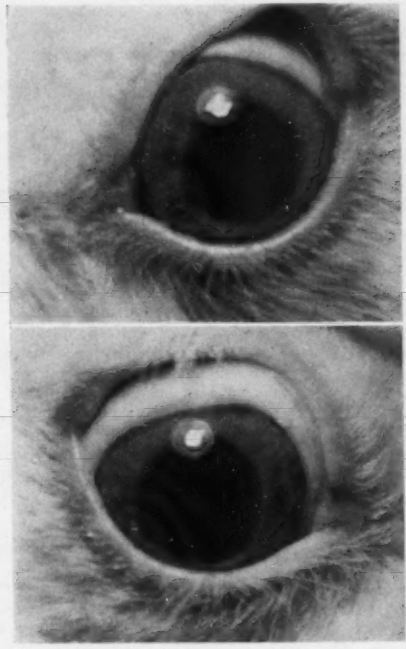

FIG. 2—Appearance of eyes seven days following challenge with Pseudomonas aeruginosa in animals given 140 mg. of Pseudomonas-immune gamma globulin forty-eight hours prior to challenge.

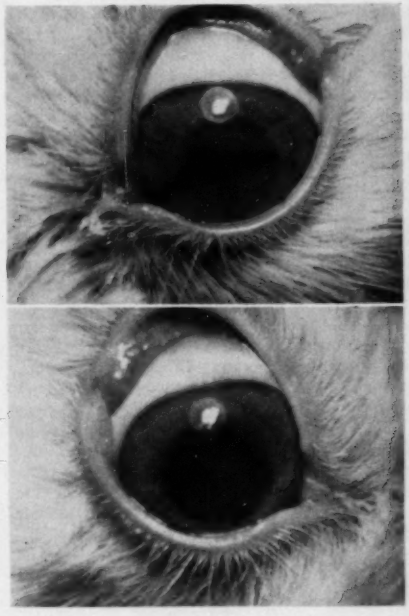

FIG. 3—Appearance of eyes seven days following challenge with Pseudomonas aeruginosa in animals given 140 mg. of Pseudomonas-immune gamma globulin twenty-four hours after challenge. Representative of fourteen out of twenty-four eyes in groups 5, 6 and 7.

monas-immune gamma globulin was given prior to challenge with the homologous organism. Pseudomonas-immune gamma globulin given six, twelve or twenty-four hours after challenge, controlled the infection in fourteen of twenty-four eyes.

It was demonstrated that intramuscularly administered immune globulin was effective in protecting the cornea, an avascular tissue, against infection.

REFERENCE

1. McMeel, J. W.; Wood, R. M., and Senterfit, L. B.: Active immunization against Pseudomonas infection of the cornea, in press.

DISCUSSION

TED SUIE, Ph.D., Columbus, Ohio (by invitation): This very interesting and well controlled work by Dr. McMeel and Dr. Wood demonstrates that the cornea, in spite of its avascularity, may participate as part of a systemic bacterial immunity both actively and passively. This research is a logical sequela to the experiments, recently reported by Fisher and Allen [A.J.O., 46:249-255 (Nov., pt. II), 1958], who found that partially purified protease from *Pseudomonas aeruginosa* can produce extensive damage to the cornea of the rabbit. They showed that this protease was antigenic by virtue of its ability to produce specific antibodies. In their discussion they also mentioned that Pseudomonas antisera would protect the cornea *in vivo* from destructive ulcerations produced by this organism. Assuming that passive immunization would be effective in human corneal ulcers due to Pseudomonas, there would be a major factor,

namely the time element, which might limit its effectiveness. We are all aware that many of the patients seek ophthalmic care only after extensive irreversible damage has occurred. Furthermore, bacteriologic confirmation requires at least twenty-four hours and antibiotic studies require additional time. Although direct smears to determine the presence of gram negative rods is always indicated, we have not always been successful in demonstrating them even though their growth on artificial media may be apparent within twenty-four hours after culturing. We know also that other morphologically similar gram-negative rods including Proteus and Escherichia may produce corneal ulcers which are indistinguishable clinically from those produced by Pseudomonas. However, if the patient is so fortunate as to be seen and to have a presumptive diagnosis made during the very early stages of corneal infection, specific antisera might prove to be of value in preventing a rapid and severe corneal destruction. The factors which would influence the effect of passive immunization would include the source of antiserum, its potency, the quantity given, the routes of administration and the ease of antibody transport through the blood-aqueous barrier. In this connection it would be very interesting and useful to determine whether the Pseudomonas antiprotease serum would protect the cornea from destruction by Pseudomonas as well as by other gram-negative rods.

I agree with the authors that antibiotics should be given concurrently with serum therapy. Here again, the usefulness of antibiotics such as polymyxin B against Pseudomonas would be measured by the extent of corneal damage at the time the therapy is initiated.

Active immunization against Pseudomonas ulcers certainly would be limited, since these infections are not associated with a specific age group, sex or occupation. Individuals with a history of recurrent attacks of infections with Pseudomonas or preoperative individuals harboring Pseudomonas as part of their normal ocular flora may benefit from such immunization. Before advising such prophylactic immunization in humans, the clinicians must consider the possible adverse effects of toxicity and hypersensitizations.

Again, I want to congratulate these authors for their stimulating research.

ACTIVE IMMUNIZATION AGAINST PSEUDOMONAS INFECTION OF THE CORNEA

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RONALD M. WOOD, PH.D.
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BY INVITATION

ONE of the most severe corneal infections encountered clinically is that caused by Pseudomonas.

A review of the literature[1-11] and the cases at the Wilmer Institute from 1939 to 1957 revealed forty-four cases of corneal ulcer due to Pseudomonas. Eleven eyes were enucleated, twenty patients had vision of 20/200 or less, and thirteen had vision better than 20/200.

This experimental study was undertaken to determine the degree of protection against infection obtainable by active immunization.

From the Wilmer Ophthalmological Institute of the Johns Hopkins Hospital and University.
This work was supported by a grant (B43-C11) from the Department of Health, Education and Welfare, Public Health Service National Institute of Health, and in part by a grant from the Knights Templar Eye Foundation.

MATERIALS AND METHODS

A freshly isolated strain of Pseudomonas aeruginosa from a human corneal ulcer was used throughout the experiment. The culture was maintained in Trypticase-soy broth (B.B.L.) enriched with 1 per cent rabbit blood, and on Trypticase-soy agar plates enriched with 3 per cent rabbit blood. The cultures were transferred at 24-hour intervals.

VACCINE PREPARATION

Four flasks, each containing 200 ml. of Eugon broth (B.B.L.), were inoculated with Pseudomonas aeruginosa, and after eighteen hours' incubation the growth was harvested by centrifugation. The organisms were then washed three times in physiological saline solution buffered to a pH of 7.2, containing 0.5 per cent phenol. The organisms were heated at 60 degrees centigrade for thirty minutes in a water bath, and standardized to a light transmittance of 50 per cent using the Coleman model B Spectrophotometer.

Immunization of Animals

Sixteen normal rabbits of mixed breed with an average weight of five pounds were used. Before beginning immunization, blood samples were taken for reference. The course of immunization consisted of intravenous injections of 0.1, 0.2, and 0.5 ml. of vaccine given on three consecutive days at weekly intervals for three weeks. Seven days after the last injection, blood samples were taken to determine antibody titers.

Challenge Inoculation

Two weeks after completion of the immunizing injections the group of sixteen immunized animals and a group of seven normal animals were challenged. The eyes were first anesthetized with 1 per cent Pontocaine, and the epithelium of each cornea was broken by a 0.2 mm. deep 5.0 mm. trephine incision. Two drops of a 24-hour broth culture of Pseudomonas was instilled into each eye.

RESULTS

None of the animals showed antibody levels before immunization. Following the course of immunization the antibody titers against Pseudomonas were 1:20 (one animal); 1:40 (five animals); 1:80 (six animals); 1:160 (three animals); and 1:320 (one animal).

Forty-eight hours after the challenge inoculation, all fourteen eyes of the seven normal control animals developed severe fulminating purulent keratoconjunctivitis with corneal ulceration. In contrast, of the thirty-two eyes in the sixteen immunized rabbits, twenty-seven were almost completely quiescent, show-

TABLE I

COMPARISON OF CORNEAL INFECTION IN IMMUNE AND NONIMMUNE RABBITS FORTY-EIGHT HOURS AFTER CHALLENGE

GROUP	NO. OF EYES	DEGREE OF INFECTION		
		MINIMAL	MODERATE	SEVERE
Normal	14	0	0	14
Pseudomonas-Immune	32	27	4	1

ing little or no involvement of the cornea. Of the remaining five eyes, four showed minimal evidence of infection and the one classed as severe was not as bad as any of the control eyes (table I).

Because of the severity of the infection in the control group, these animals were sacrificed at this time. The immunized group was observed for an additional seven days. At the end of this time only two of the sixteen animals showed residual scarring.

SUMMARY

The corneas of rabbits immunized with Pseudomonas aeruginosa vaccine resisted challenge infections with the homologous organism. Of thirty-two eyes, twenty-seven were completely protected, four showed moderate infection, and one, severe infection. Of fourteen control eyes in normal rabbits, all became severely infected.

CONCLUSION

Circulating antibody against Pseudomonas, induced by a course of active immunization with a vaccine, gave significant protection against subsequent corneal challenge with the homologous organism.

REFERENCES

1. Ainslie, D.: Treatment of corneal infection with *Ps. pyocyanea* by subconjunctival injection of polymyxin E, Brit. J. Ophth., 37:336-342 (June) 1953.
2. Bignell, John L.: Infection of the cornea with *B. pyocyaneus;* clinical study and summary of ten cases personally observed, Brit. J. Ophth., 35:419-423 (July) 1951.
3. Eareckson, Vincent O.; Miller, Joseph M., and Long, Perrin H.: Infection of eye to Pseudomonas aeruginosa treated with polymyxin B and "Varidase," A.M.A. Arch. Ophth., 49:158-160 (Feb.) 1953.
4. Fraser, H.: Subconjunctival injection of polymyxin B, Brit. J. Ophth., 37:369 (June) 1953.
5. Lepard, C. W.: B. pyocyaneus ulcer. Report of three cases: Results of sulfapyridine therapy in one case, Tr. Am. Acad. Ophth. & Otol., 46:55-60 (Sept.-Oct). 1941.
6. MacDonald, Marguerite: *Ps. pyocyaneus* eye infection, Brit. J. Ophth., 37:370-373 (June) 1953.
7. Maschler, J.: A case of pyocyaneus ring abscess of the cornea treated with streptomycin, Brit. J. Ophth., 32:426-428 (July) 1948.
8. Moorman, Lemuel T., and Harbert, Fred: Treatment of pseudomonas corneal ulcers, A.M.A. Arch. Ophth., 53:345-350 (March) 1955.
9. Pendexter, S. E., Jr.: B. pyocyaneus corneal ulcer treated with pencillin, Am. J. Ophth., 31:862-863 (July) 1948.
10. Wadensten, L.: A case of pyocyaneus keratitis treated with aureomycin, Acta Ophth., 32:483-484, 1954.
11. Williams, Richard Kennon; Hench, Miles E., and Guerry, DuPont, III.: Pyocyaneus ulcer, Am. J. Ophth., 37:538-544 (April) 1954.

RECENT ADVANCES IN ASEPTIC SURGICAL TECHNIQUE

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THE incidence of endophthalmitis after cataract surgery has been reduced during the past century about fifty-fold. According to Fuchs,[9] between 4 and 10 per cent of all eyes operated upon for cataract were lost before the introduction of Listerian antiseptic technique. A series of 60,000 cataracts reported during the past twenty years showed an incidence of 1.6 infections per thousand cases.[3] If unreported cases are included, the modern incidence is probably about two infections per thousand operations. Accurate data on the number of cataract operations performed annually in the United States and Canada are impossible to obtain. If the figure be estimated at 50,000, it is possible that 50 to 100 eyes are destroyed annually by bacterial infection following cataract surgery alone. A few more eyes are lost from bacterial endophthalmitis following surgical procedures for retinal detachment and glaucoma.

Not only bacterial but also fungus infections may be contracted during intraocular operations. Fine and Zimmerman,[8] in a recent contribution to the etiology of uveitis and endophthalmitis, have described the clinical picture of intraocular fungus infection and have shown that this may be delayed for weeks or months after the operation in which fungi were introduced into the eye. The number of eyes lost from such infections cannot be accurately estimated. Any eye which develops delayed postoperative inflammation characterized by dense white exudates in the anterior vitreous should be suspected of harboring a fungus infection.

So far as is known, a viral etiology has not been established for any case of postoperative endophthalmitis.

The etiology of some of the bacterial infections which have been reported is shown in table I. Two fifths of all reported infections have yielded *Staphylococcus aureus* on culture. Gram-negative bacilli have accounted for another third of all cases in which the cause has been established, and with still other cases of presumed infection, reports on cultures have indicated no growth.

Attempts to trace the source of bacteria causing postoperative endophthalmitis have been unrewarding in most instances. This is probably because the search has been directed chiefly toward objects and materials still available two or more days after the operation was performed. In some of these instances, however, recovery of the same species from infected eyes and from solutions which were used before or during operation has left little doubt of the role of contaminated solutions. Table II shows some disastrous instances of this type of contamination.

Apart from such obvious sources, there is evidence that either the patient or the surgeon may contribute the in-

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TABLE I

ETIOLOGY OF POSTCATARACT ENDOPHTHALMITIS[5]

ORGANISM	INFECTION	PER CENT
Gram-positive		
Staph. aureus	26	42.0
Pneumococcus	6	9.7
Streptococcus (all)	6	9.7
Gram-negative		
Pseudomonas	15	24.1
A. aerogenes	4	6.5
E. coli	3	4.8
Proteus sp.	1	1.6
N. catarrhalis	1	1.6
Totals	62	100

TABLE II

PSEUDOMONAS INFECTION FROM CONTAMINATED SOLUTIONS

SOLUTION	WHERE USED	HOW USED	EYES LOST
Benzalkonium chloride	Hospital OR	Instruments	3
Benzalkonium chloride	Hospital OR	Skin antiseptic	5
Saline	Hospital OR	Anterior chamber	6
Sulfonamide	Industrial dispensary	Conjunctival instillation	5

fectious agent at the time of the operation. This evidence, in respect to the patient's contribution, is as follows: Surveys have invariably shown that pathogenic bacteria are present on a certain percentage of clinically normal conjunctivas and lid margins. In a series of 5,000 eyes cultured preoperatively at two London hospitals, Smith[13] found coagulase-positive staphylococci in 8 per cent, and other potential pathogens in another 7 per cent of cases. In a series of 1,004 preoperative cultures, Cason and Winkler[14] found potential pathogens in 12 per cent. Moreover, they were able to correlate postoperative infection with preoperative conjunctival cultures in four of twelve infections. Using a new quantitative technique which reveals not only the species but also the relative numbers of bacteria present during an operation, we have

recovered coagulase-positive staphylococci from swabs used in 9 of 36 operations studied to date.[3] Our experience is that up to 200 pathogenic bacteria (coagulase-positive staphylococci) may be present during an operation without having the slightest effect on the postoperative course of an eye. Nothing is known about immunologic relationships between host and parasite which might prevent or predispose to the development of clinical infection when such pathogens enter the anterior chamber.

A factor of undetermined significance may lie in our finding that the incidence of postoperative infections was eight times greater in second eyes operated upon during the same hospital admission as their uninfected fellow eyes.

An argument in favor of intracapsular cataract extraction is the fact that in a series of 10,000 consecutive cataract operations at the Massachusetts Eye and Ear Infirmary[3] the incidence of postoperative endophthalmitis (excluding presumptive cases of lens-induced uveitis) was eight times greater after extracapsular extraction, intentional or accidental, than after intracapsular extraction. Here a seemingly valid explanation lies in the fact that lens material is an excellent culture medium as compared with aqueous or vitreous humor.

Finally, we have evidence that bacteria derived from the upper respiratory tract of a member of the surgical team may find their way into the anterior chamber and there set up an infection.

If these observations may be accepted as valid and significant, the following objectives are worth attaining: (1) elimination of pathogenic bacteria from the immediate environment of an intraocular procedure; (2) causing a minimum trauma to the eye during the operation. We are presently concerned only with the first of these objectives. The second is assiduously cultivated by the majority of ophthalmic surgeons, whose preference for the intracapsular operation is almost unanimous.

A systematic approach to elimination of bacterial pathogens from the local environment should be based on the following points:

1. Elimination of pathogens from the patient's conjunctival sac and lid margins
2. Absolute sterility of all objects and medications used at operation
3. Strict discipline in aseptic technique by doctors and nurses with emphasis on proper masking and the elimination of unnecessary conversation in the operating room
4. Adequate ventilation at the rate of not less than 12 air changes per hour, using air filtered through deep-bed glass fiber media

For sterilization of the conjunctival sac, there are no studies which establish the superiority of any one antibiotic or combination of antibiotics. We are currently performing a long-term study which may permit significant conclusions as to the relative effectiveness of chloramphenicol and neomycin topical solutions in preventing postoperative infection. Any bacteriologic evaluation of antibiotics in vivo must be controlled in respect to carry-over of bacteriostatic concentrations of antibiotics into culture media.

For degerming the skin of the lids and brow, we favor a two-minute scrub with green soap followed by a two-minute rub with isopropanol, 70 per cent. Evidence for the superiority of this regime has been furnished by Blank and Coolidge.[5] No amount of scrubbing or alcohol will eliminate staphylococci from the deep glands of the skin and lid mar-

gins. The wary clinician will be on the alert for crusting, loss of lashes and other signs of chronic staphylococcal blepharitis.

The principal advances to be noted in the field of instrument sterilization are the increased use of dry heat and of the newer gaseous sterilants. At the Massachusetts Eye and Ear Infirmary, the preferred practice is to sterilize entire kits of instruments in the hot air oven overnight. Separately packaged eye knives and other sharp instruments sterilized by dry heat are available for immediate use at all times. Stainless steel instruments can either be processed in advance by dry heat or can be autoclaved in the usual manner.

Instruments and optical equipment which will not withstand hot air or steam sterilization can be sterilized by one of the gaseous sterilants, formaldehyde, ethylene oxide or beta-propiolactone. We have abandoned the use of formaldehyde because of its tendency to polymerize on surfaces and because of its persistent, pungent odor. Ethylene oxide has excellent penetrating power but is explosive and flammable in the presence of air. It takes several hours to sterilize even when it is used with complete control of temperature, gas concentration and humidity. Equipment providing automatic control of all these factors costs several thousand dollars. Skeehan, King and Kaye[12] have described the use of ethylene oxide in combination with Freon 12 in a converted autoclave shell. The experience we have had with ethylene oxide over a period of several years indicates that this gas is effective only in the hands of trained personnel, and that even under carefully controlled conditions the incidence of "skips" or failures is annoyingly frequent.

A new compound of great promise in the field of cold or gaseous sterilization is beta-propiolactone (BPL), whose properties as a sterilant were discovered during extensive screening of compounds at Ft. Detrick, Maryland. A liquid at room temperature, BPL boils at 155 to 160 degrees C. and vaporizes fairly rapidly at lower temperatures. It is convenient to handle, with simple precautions against inhaling the vapor or spilling the liquid on the skin or in the eye. Its toxicity under these circumstances is comparable to that of strong alkalies. In water it hydrolyzes to nontoxic products.

The great advantages of beta-propiolactone are its rapidity of action and the fact that no special equipment and very little training are required for its use as a gaseous sterilant. In the vapor phase, a concentration of 13 milligrams per liter of space will kill the most resistant spores in sixty minutes.[4] For example, a medical microscope can be sterilized under a 16-liter plastic or metal wastebasket in the presence of a few drops of water on one paper towel and 0.18 cc. of BPL dispersed on another paper towel. Vaporization of BPL can be effected by atomization, by heating the liquid in an air stream, or by drawing a partial vacuum in a hermetically sealed container. BPL has been used to sterilize the interior of a two-story building,[10] and there is almost no limit to the size of spaces and their contents which can be sterilized with it. In ophthalmology, BPL sterilization can be applied to ophthalmoscopes, slit lamps, oscillating saws and other bulky objects. Although instruments can be rapidly sterilized by immersion in freshly prepared aqueous solutions of BPL, provided they are thoroughly rinsed in sterile water immediately thereafter, we do not recommend the use of BPL for routine instrument sterilization.

Most dangerous are the solutions and medications which enter the anterior

chamber during surgical procedures. Chief among these is the irrigating fluid —normal saline solution or combined electrolytes. We use only sealed units no larger than 500 cc. which have been sterilized in the final container. Commercial preparations intended for intravenous use offer a reasonable guarantee of security and have always proved sterile in our tests. No hospital should undertake to put up or sterilize intraocular irrigant solutions unless it is prepared to assume full responsibility for the pharmacologic and bacteriologic purity of the final product. All ophthalmic surgeons should personally investigate the source and method of preparation of fluids for intraocular use in their hospitals. I believe that every ophthalmologist has the right to see a negative culture report of any lot of intraocular irrigant he is to use. The same applies to other medications used in the operating room. The sterile disposable dose-unit is to be preferred whenever an eye is injured or open, even though the injury may be no more than a corneal abrasion.[11]

Although the role of air-borne infection in ophthalmic and general surgery has not been fully elucidated, there is strong evidence that bacteria from the upper respiratory tract of a member of the surgical team can produce postoperative intraocular infection.[3] Possibly this mechanism operates more often than has been generally believed. The frequency of coagulase-positive staphylococci in the anterior nares of general and hospital populations is well established.[7]

When this circumstance is combined with the habit of many surgeons of conversing freely while wearing masks of low antibacterial efficiency, it is not surprising that infectious droplets may find their way into the conjunctival sac and thence into the anterior chamber. Surgical silence and the adoption of more efficient masks[1] are two obvious solutions to this problem.

The importance of adequate ventilation and the means of obtaining a supply of sterile air in the operating room have been discussed elsewhere.[2]

In summary, it may be said that antibiotics have reduced but have not eliminated the possibility of postoperative intraocular infections. The intracapsular technique is undoubtedly responsible for a lowering of the infection rate. Retention of lens material within the eye suggests the need for subconjunctival or systemic antibiotics at the end of the operation. Absolute sterility of all instruments and medications used in surgery is essential. A greater awareness of bacteria in the surgical environment is needed if sporadic cases of postoperative endophthalmitis are to be avoided.

REFERENCES

1. Adams, Ralph, et al: Control of infections within hospitals, J.A.M.A., 169:1557-1567 (April 4) 1959.

2. Allen, Henry F.: Air hygiene for hospitals. II. Efficiency of fibrous filters against staphylococcic droplet nuclei and bacteria-bearing dust, J.A.M.A., 170:261-267 (May 16) 1959.

3. Allen, Henry F., and Mangiaracine, A. B.: Factors predisposing to bacterial endophthalmitis after cataract surgery. To be published.

4. Allen, Henry F., and Murphy, J. T.: Sterilization of instruments and materials with beta-propiolactone. To be published.

5. Blank, Irvin H., and Coolidge, Mary H.: Degerming the cutaneous surface. I. Quaternary ammonium compounds. II. Hexachlorophene (G-11), J. Invest. Dermat., 15:249-263 (Sept.) 1950.

6. Cason, Louise, and Winkler, C. H.: Bacteriology of the eye. I. Normal flora,

A.M.A. Arch. Ophth., 51:196-199 (Feb.) 1954.

7. Clarke, Suzanne K. R.: Nasal carriage of Staphylococcus aureus, J. Path. & Bact., 73:253-259, 1957.

8. Fine, Ben S., and Zimmerman, Lorenz E.: Exogenous intraocular fungus infections: With particular reference to complications of intraocular surgery, Am. J. Ophth., 48:151-165 (Aug.) 1959.

9. Fuchs, Ernst: Memories of Vienna clinics, Am. J. Ophth., 9:605-607, 1926.

10. Hoffman, R. K., and Warshowsky, B.: *Beta*-propiolactone vapor as a disinfectant, Applied Microbiology, 6:358-362 (Sept.) 1958.

11. Riegelman, Sidney, and Vaughan, Daniel G.: A rational basis for the preparation of ophthalmic solutions, J. Am. Pharm. A. (Practical Pharmacy Edition) 19:474, 1958.

12. Skeehan, R. A., Jr.; King, J. H., Jr., and Kaye, Saul: Ethylene-oxide sterilization in ophthalmology, Am. J. Ophth., 42:424-430 (Sept.) 1956.

13. Smith, C. H.: Bacteriology of the healthy conjunctiva, Brit. J. Ophth., 38:719-726, 1954.

14. Winkler, C. H., and Cason, Louise: Bacteriology of the eye. II. Role of gram-negative bacilli in infections following cataract extraction, A.M.A. Arch. Ophth., 51:200-203 (Feb.) 1954.

DISCUSSION

JAMES H. ALLEN, M.D., New Orleans: I wish to congratulate Dr. Allen on his excellent paper calling our attention to several important factors which are too frequently taken for granted these days.

One of the most important steps in the development of modern surgery was the advance from the techniques of antiseptic surgery to those of aseptic surgery.

Dr. Allen has given us four points which should serve as a guide in the maintenance of asepsis in ocular surgery. The first of these is the elimination of pathogenic organisms from the patient's conjunctival sac and lid margins. This means freedom from inflammatory disease of the eye and adnexa, especially the conjunctiva, lacrimal sac, lids and lid margins; it also means the absence of pathogenic organisms in cultures made from the conjunctiva and lid margins just prior to surgical procedures. To me, the use of antibiotics prophylactically in this situation in lieu of negative cultures is a reversion to antiseptic surgery and is to be deplored.

I am very happy that Dr. Allen is using soap and alcohol for preparation of the skin. These preparations are much more effective than recent substitutes. However, it is important to emphasize that 70 per cent alcohol should be used for this, as it is the most bactericidal concentration. Either stronger or weaker concentrations of alcohol are less bactericidal. Furthermore, allowing the alcohol to evaporate rather than blotting and drying it off with sponges increases its bactericidal effectiveness.

The second of Dr. Allen's points is absolute sterility of all objects used at operation. I believe that Dr. Allen has adequately emphasized the absolute necessity of sterile solutions and medications. But I should like further to emphasize that at present there is no substitute for heat (dry heat or autoclave) sterilization of surgical instruments. Gas sterilization, chemical sterilization and irradiation sterilization with ultraviolet all have their places in other areas, such as sterilization of rooms and major optical equipment, but at present I do not believe that they have a place in the sterilization of the instruments used for intraocular surgery.

The third point that he gives us is strict discipline in aseptic techniques by operating room personnel. This point cannot be overemphasized. A slight error or slip in technique may make the difference between the saving or the losing of an eye.

The fourth point, adequate ventilation, is important in the operating room, but the air must be properly filtered, and it must be controlled to prevent drafts from blowing over the operative site and the instrument table. Incidentally, one must keep in mind the necessity of replacement of the filters in air conditioning systems at frequent intervals.

Finally, it should be emphasized that the eye, like the central nervous system, may be affected and damaged by dust-borne organisms, such as the Bacillus subtilis, which are not pathogenic elsewhere in the body. The ocular surgeon and his team must therefore be even more vigilant in the maintenance of aseptic techniques than most other surgeons.

ADOLPH POSNER, M.D., New York: I wish to congratulate Dr. Allen on his significant contribution to the problem of asepsis. I was glad to hear that Dr. Allen is opposed to the use of glove powders and that they are not being used at the Massachusetts Eye and Ear Infirmary; yet most hospitals today are still using glove powders that are prepared from cornstarch.

It is important to check carefully any sterilization procedure in order to be sure that we kill the many spores of bacteria and fungi which these powders contain. I believe we cannot emphasize too strongly that starch-derivative absorbable glove powders may act as culture media, just as Dr. Allen has mentioned. This powder is a particularly good culture medium for fungi which, according to several recent reports, cause serious endophthalmitis after cataract extraction.

The fact that starch-derivative powder can support life was shown in an experiment performed at the Manhattan Eye, Ear and Throat Hospital with the assistance of Richard Inglima. Sterile starch powder was placed in the bottom of six test tubes and sterile talcum powder was put in another six tubes. Of each series, five tubes were inoculated with Penicillium spores and the other tubes were used as controls. After eleven days, a sample of air from each tube was cultured on Sabouraud's medium. Sixty-eight colonies of Penicillium were recovered from the air in the tubes containing starch powder, whereas the air from the talcum tubes yielded only three colonies. Just as the air in our test tubes was contaminated, so does the air in the operating room become contaminated by the use of glove powder. This is especially true of the starch-derivative powder, which is highly dispersible and acts as a culture medium.

The fact that starch-derivative glove powders are still being used in most hospitals points up the general unawareness of the threat to asepsis from the easily contaminated residue left in the operating room. It would seem that the role of antibiotics in the development of resistant strains of organisms has been so dramatic as to divert attention from the new glove powder which was introduced some ten years ago, about the same time as antibiotics came into widespread use. I believe that the role of the glove powder residue left in operating rooms deserves careful investigation as a source of air contamination and that it may be responsible for many hospital infections.

SEPTAL DERMOPLASTY FOR CONTROL OF NOSEBLEEDS CAUSED BY HEREDITARY HEMORRHAGIC TELANGIECTASIA OR SEPTAL PERFORATIONS

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IN 1901, William Osler published a classic paper called "On a Family Form of Recurring Epistaxis, Associated with Multiple Telangiectases of the Skin and Mucous Membranes." He reported the histories of three patients. Here is an excerpt from his paper describing a man who had suffered nosebleeds all his life:

> When ten years old the patient began to have epistaxis which often followed the trick of walking on his hands. He would bleed quite profusely for part of a day, or for some hours every day or two for ten days or more, until he got quite weak and anemic.
>
> In the first ten days in the hospital there were six bleedings from the nose. Cauterization of the angiomata on the septum was followed by quite profuse hemorrhage which was readily stopped. The next day the hemorrhage recurred and he lost 580 cc. of blood before it was checked by plugging. Half an hour later he had a second hemorrhage in which 820 cc. were lost. He said he frequently had much more profuse hemorrhage.

The severe epistaxis suffered by Osler's patient is typical of the nosebleeds of many patients with hereditary hemorrhagic telangiectasia (Rendu-Osler-Weber disease). In some, epistaxis is a daily occurrence, profuse enough to cause severe anemia. In others, there may be an occasional mild nosebleed and then, once or twice a year, an exsanguinating hemorrhage. Besides epistaxis, there may be serious bleeding from the gastrointestinal tract. In fact, any epithelial surface may bleed, and there may be hemorrhage into any organ. After epistaxis and gastrointestinal bleeding, hemorrhages from the lips, tongue and gums are most common. Rarely, hematuria, hemothorax, or bleeding into the spleen, liver or meninges occurs.

Although the telangiectases are widespread, in virtually every patient epistaxis is the cardinal symptom; if it can be controlled, there is usually no serious problem. As one of Osler's patients put it, "I have been in the habit of bleeding to death every day."

The Telangiectasia

The telangiectatic spots that bleed so severely are easily seen in all parts of the nasal mucosa. Other common sites are the lips, the tongue, the palate, the cheeks, and the fingertips. The lesions are red and slightly raised. They blanch when compressed under a glass slide.

Microscopically, these telangiectases are thin-walled vessels without elastic or muscular tissue. They look like vascular sinuses interposed between arteries and veins. When traumatized, some telangiectases spurt like an artery, others bleed more like a vein. Because of their fragile walls and superficial location, they bleed with every minor trauma. Absence of contractile elements in the vessel walls make it difficult for bleeding to stop spontaneously.

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SEPTAL DERMOPLASTY IN HEREDITARY HEMORRHAGIC TELANGIECTASIA

Septal dermoplasty is a new surgical method of correcting the severe nosebleeds that result from hereditary hemorrhagic telangiectasia. The same procedure may be used to close septal perforations, but because the techniques differ slightly, and the results considerably, the closure of septal perforations will be discussed later.

In the past, such surgical measures as cautery, packing, arterial ligation, and injection of sclerosing solutions have been largely ineffective in controlling the nosebleeds of patients with hereditary hemorrhagic telangiectasia. Similarly, medical measures, such as the use of drugs to reduce capillary fragility, have been ineffective. Both surgical and medical measures were ineffective because they did nothing to make the mucous membrane more resistant to trauma.

Principles

1. In hereditary hemorrhagic telangiectasia, although all of the nasal mucosa shows telangiectasia, only the anterior part of the nasal septum bleeds regularly. Other areas with the same telangiectasia seldom bleed, because they are less exposed to trauma. Sometimes a prominent telangiectasia on the anterior end of the inferior turbinate becomes traumatized and bleeds.

2. Replacement of the fragile mucous membrane of the anterior third of the septum with skin provides a tough epithelium where trauma occurs.

The Technique

1. *Premedication.* Ninety minutes before the operation, the patient swallows two capsules of Nembutal (0.1 gm. each). Thirty minutes before the operation, he receives 100 mg. Demerol intramuscularly. The barbiturate is especially important because relatively large amounts of local anesthetic are used in the operation, and barbiturates are specific safeguards against toxic effects of local anesthetics.

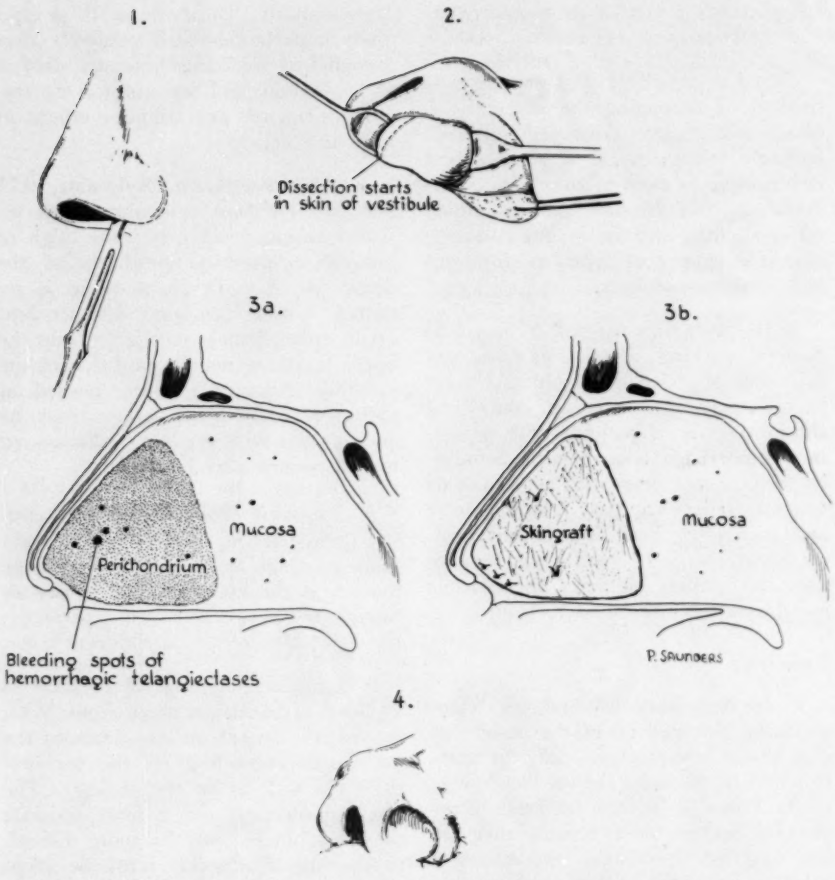
2. *The Anesthesia.* Xylocaine, 0.75 per cent (without epinephrine), is injected subcutaneously into the thigh or buttock to produce anesthesia of the donor site. Usually about 40 cc. is required. Later, Xylocaine, 1.0 per cent (with epinephrine), is injected into the upper lip, the columella, and skin of the vestibule. About 3.0 cc. are needed on each side. The nasal septum may be anesthetized with the same solution, or topical cocaine may be used.

3. *The Skin Graft.* The Brown electric dermatome is used to remove skin from the thigh or buttock. Probably the buttock is the better place because, although the donor site heals perfectly, the new skin remains a different color.

A dermatome setting of 20/1000 to 25/1000 of an inch is about right. With the electric dermatome, the depth of the cut varies depending on the pressure used (as well as on the setting). The Paget dermatome cuts a more accurate thickness but is generally more difficult to operate. The graft must be large enough to cover the anterior third of *both sides* of the nasal septum.

After the graft is cut, the donor site is covered with petrolatum gauze and then several layers of dry gauze. A nurse bandages the donor site, and the patient turns over for the nasal operation. The graft is kept moist until needed.

4. *The Nasolabial Incision.* An incision (fig. 1) in the nasolabial sulcus separates the ala from the upper lip. The incision should extend a little into the floor of the nose. Bleeding is neg-



FIGS. 1 to 4—Septal dermoplasty as used in patients with hereditary hemorrhagic telangiectasia. Note in 3-A and 3-B that while the graft need not cover more than the anterior third of the septum, it must reach all the way from the dorsum to the floor.

ligible. A single suture through the cut edge of the ala serves as traction.

5. *Excision of Septal Mucosa.* The next incision (fig. 2) is made in the skin of the vestibule just anterior to the mucocutaneous junction. In other words, a little skin is excised along with the septal mucosa. The incision is continued to the floor of the nose and upward to the top of the septum. An assistant retracts the upper lateral nasal cartilage. Perichondrium is spared; only mucosa is excised.

Scissors or knife is used to dissect the mucosa anteriorly. Further back, where these instruments are difficult to use, a curette works better.

The exact technique matters little. The principle is to excise mucosa and leave

perichondrium (fig. 3-A). If some cartilage or bone is exposed, no harm is done. However, all particles of mucosa should be removed so that, when the skin graft is applied, small pieces of mucus-secreting epithelium will not be entrapped.

Hemostasis is no problem. Bleeding is controlled with cotton soaked in topical Adrenalin. Electrocautery is occasionally needed to stop persistent bleeding from arterial vessels.

The same procedure is followed on each side.

6. *Application of the Graft.* The graft is cut in half; one piece will be applied to each side of the septum (fig. 3-B). First, the graft is fixed to the cut edge of the vestibular skin with sutures of 4-0 plain catgut. This step makes the graft easier to control. Once it is fixed anteriorly, the graft can be pushed back into the nose and measured against the bare area. Usually the graft is a little too large, but it does not matter because any excess skin eventually sloughs. On the other hand, if a raw area is left uncovered, it tends to form granulations that bleed. It is *very important* to make certain that the graft reaches all the way from the *top* of the septum to the *floor* of the nose, especially anteriorly. Failure to cover the entire septum *anteriorly* will leave telangiectases exposed to trauma.

Once the graft is trimmed and both sides of the septum covered, two or three mattress sutures fix the central and posterior parts of the graft. A large curved cutting needle with 4-0 plain catgut is used. Finally, cotton impregnated with terramycin or bacitracin ointment is fashioned as a light packing. Firm packing is wrong because it keeps blood from entering and nourishing the graft.

The nose and upper lip are reapproximated with 5-0 Dermalon sutures (fig. 4), and the operation is finished.

Postoperative Care

Immediate Care. Pain is minimal and readily controlled with codeine.

The nasal packing is left in place four days. When it is removed, the outer layer of the skin separates from the rest of the graft; often serum has collected between the two. The graft is firmly attached to the septum except at the posterior edge, where there is a little excess skin that will eventually slough. The catgut sutures are left in place. The Dermalon sutures are removed after one week.

The donor site is not disturbed except to change the outer dressing as necessary. In two weeks the inner petrolatum gauze may be removed. Usually the leg is healed.

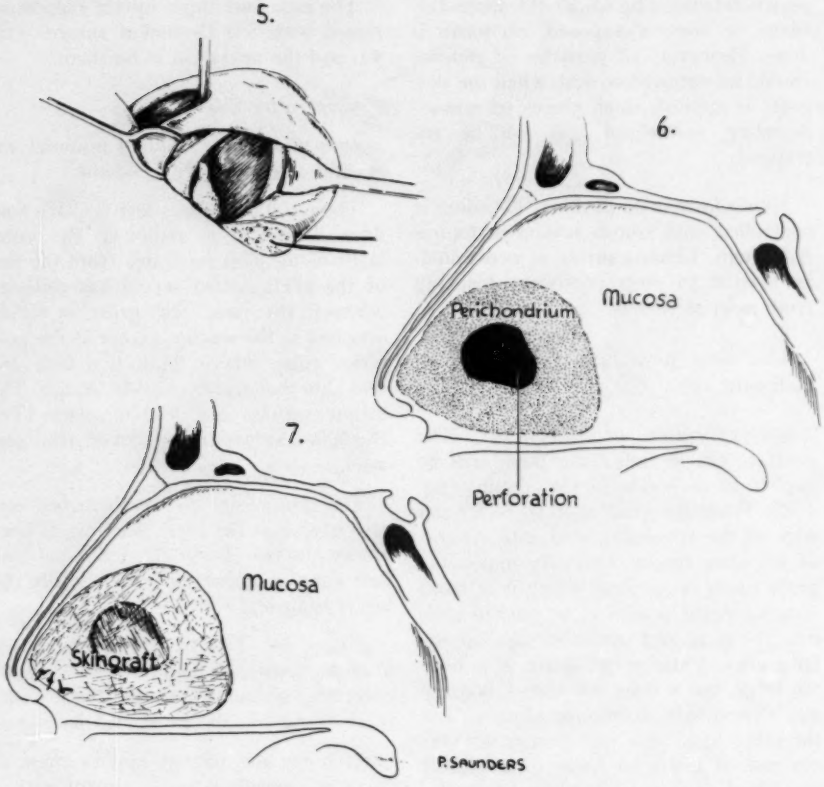
Late Care. The patient is seen once a week. Crusts are removed, excess skin carefully trimmed, and antibiotic ointment applied to the graft and its edges.

At home the patient applies mineral oil or an aquaphor-type ointment with a cotton-tipped applicator that has been wound so as to leave a tuft at the end. Ordinary commercial applicators will not do; they are too thick and their ends are too firm.

The nose continues to crust for several weeks. Eventually the crusting stops altogether, or almost so. Some patients may wish to clean the graft once a day with a bland ointment and cotton applicator.

Results

Seven patients with hereditary hemorrhagic telangiectasia have been operated upon in the past year. There were four women and three men, only two of



FIGS. 5 to 7—Septal dermoplasty as used in patients with septal perforations that bleed or crust.

whom were related. The oldest patient was 77 years old and the youngest 30.

The first operation was done one year ago. The operations on the other six patients were all done at least four months ago. None of the patients have had a nosebleed of more than a few drops since the operation. These few drops of blood have come from telangiectases on the anterior ends of the inferior turbinate and not from the septum. Patients are told that no effort will be made to remove all intranasal telangiectases. *They are told to expect an occasional minor nosebleed caused by trauma to one of the remaining telangiectases.*

SEPTAL DERMOPLASTY AND SEPTAL PERFORATIONS

Ordinary septal perforations, the results of nasal operations, nose picking or infection, often cause no symptoms (fig. 5), but sometimes the margins of the perforations bleed and crust. Although such nosebleeds are usually not serious, they may be frequent enough to be troublesome.

Septal dermoplasty is not intended for closure of asymptomatic septal perfora-

tions. Patients with asymptomatic perforations are often unaware of their abnormality. But when bleeding or crusting is a major problem, the operation is worthwhile.

The Operation

Septal dermoplasty, when used in the closure of septal perforations, differs a little from the same operation when used in patients with hereditary hemorrhagic telangiectasia. First, the graft must be thicker. Instead of a dermatome setting of 20/1000 to 25/1000 of an inch, 32/1000 to 35/1000 is required. Second, enough normal mucosa must be removed from around the perforation to provide a good base for the graft (fig. 6). Presumably the more raw surface, the better. In a large perforation this may mean that a considerably larger piece of skin is grafted (fig. 7) than is necessary in patients with hereditary hemorrhagic telangiectasia. Finally, the packing must be very *loosely* applied, particularly over the area of the perforation. Too much pressure keeps blood from entering the graft.

Postoperative Results

Of six patients who underwent dermoplasty to correct bleeding septal perforations, two have recurrent perforations. Both of these patients had perforations extending almost the height of the septum. A third patient with an equally large perforation and three other patients with smaller holes have maintained their grafts intact.

When a skin graft is used to control bleeding in patients with hereditary hemorrhagic telangiectasia, postoperative crusting is minimal or absent. In patients with septal perforations, the story is sometimes different. Skin lines the margins of the perforation (whether or not the graft again perforates), so nosebleed is controlled. However, crusting may be severe if the graft breaks down. Then the patient may have to use intranasal lubricants daily. On the other hand, in those patients in whom the grafts have remained intact, crusting has not been much of a problem, although, in general, large grafts crust more than small ones.

Prevention of perforation of the new grafts is a problem of blood supply to the graft. I think that too much packing has caused the failures. In future operations, little or no packing opposite the perforation will be used.

CONCLUSION

Skin, grafted to each side of the anterior part of the nasal septum, is very effective in controlling nosebleeds in patients with hereditary hemorrhagic telangiectasia. The same technique can be used to close certain septal perforations which cause bleeding and crusting.

REFERENCES

1. Babington, B. G.: Hereditary epistaxis, Lancet, *2*:362-363, 1865.
2. Chauffard, M. A.: Hémophilie avec stigmates télangiectasiques, Bull. et mém. Soc. méd. hôp. Paris, 13:352-358, (Serie 3), 1896.
3. Chiari, O.: Erfahrungen auf dem Gebiete der Hals und NasenKrankheiten nach den Ergebnissen des Ambulatoriums, pp. 60 et seq., Leipzig & Wien., Toeplitz & Deuticke, 1887.
4. Hanes, Frederic M.: Multiple hereditary telangiectases causing hemorrhage (Hereditary hemorrhagic telangiectasia), Bull. Johns Hopkins Hosp., 20:63-73 (March) 1909.
5. Kelly, A. Brown: Multiple telangiectases of the skin and mucous membranes of the nose and mouth, Glasgow M. J., 65:19-24 (June) 1906.
6. Legg, Wickham: A case of haemophilia complicated with multiple naevi, Lancet, *2*:856, 1876.

7. Livingston, Shields O., and Carr, Robert E.: Hereditary hemorrhagic telangiectasia: report of a case with hemothorax, J. Thoracic Surg., 31:497-503, 1956.

8. Osler, William: On a family form of recurring epistaxis with multiple telangiectases of the skin and mucous membrane, Bull. Johns Hopkins Hosp., 12:333-337 (Nov.) 1901.

9. ———: On multiple hereditary telangiectases with recurring haemorrhages, Quart. J. Med., 1:53-58 (Oct.) 1907 (Plates II).

10. Rendu: Épistaxis répétées chez un sujet porteur de petits angiomes cutanés et muqueux, Bull. et mém. Soc. méd. hôp. Paris, 13:731-733, 1896.

11. Snyder, Laurence H., and Doan, Charles A.: Studies in human inheritance. XXV. Is the homozygous form of multiple telangiectasia lethal?, J. Lab. & Clin. Med., 29:1211-1216 (Dec.) 1944.

12. Weber, F. Parkes: Multiple hereditary developmental angiomata (telangiectases) of the skin and mucous membranes associated with recurring hemorrhages, Lancet, 2:160-162 (July 20) 1907.

13. ———: Letter to the editor, Hereditary haemorrhagic telangiectasis, Brit. M. J., 1:569, 1945.

DISCUSSION

OSCAR J. BECKER, M.D., Chicago: Dr. Saunders is to be congratulated on his excellent presentation. All too frequently in cases of severe recurrent epistaxis we are content to use cautery and nasal packing, a method which results in high morbidity. That a definitive treatment is now offered is due to the efforts of Dr. Saunders. The old adage of Mark Twain's, "Everybody complains about the weather, but nobody does anything about it," certainly does not apply to him.

Patients with Rendu-Osler-Weber's disease must suffer extreme anxiety as they await their next bout with epistaxis, which may be almost exsanguinating, requiring prolonged hospitalization, blood transfusions, and the discomfort of repeated nasal packing. Certainly we must offer something more than palliation and emergency therapy. As otolaryngologists, nasal bleeding in patients is to us a paramount problem.

For a skin graft to "take" inside the nose, I suggest a thin split thickness graft, rather than a thick graft as suggested by Dr. Saunders. The thinner the graft, the more successful the healing. Also, it would eliminate the possibility of hair growth on the graft. I would also suggest a firm pack against the graft rather than a loose pack. A firm pack is essential to prevent hemorrhage or hematoma under the graft. Blood under the graft would destroy the chances of a "take."

In correcting septal perforations, I have successfully closed quite a few with a composite graft of upper lateral cartilage, sections of the anterior border of the septum with skin and mucosa attached and sections of alar cartilage (skin attached). A combination of these grafts may be used to close the perforation. The largest perforation I have closed to date was 2.5 centimeters wide. There is a limitation in the amount of upper lateral cartilage or lower cartilage which may be removed. The use of a composite postauricular graft also is of value.

In view of Dr. Saunder's success with dermoplasty for septal perforations, a composite graft from the postauricular area, which includes cartilage as well as skin would result in a firmer, more stable closure. An alternate, if available, would be to use a thin sliver of septal cartilage placed between the membranes before applying the split thickness graft.

Again, I would like to repeat, Dr. Saunders has made a real contribution to a long neglected problem in nasal surgery and nasal pathology.

HEAD AND NECK MANIFESTATIONS OF THE CERVICO-OCCIPITAL SYNDROME

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THE otolaryngologist is frequently confronted with a symptom complex about the head and neck which until recently has been little understood. Consequently, treatment has been inadequate or neglected entirely. The absence of positive neurological signs, unless the disease is extensive or of long standing, is another reason why this condition is often overlooked. Most of the material on the subject has been written by internists, neurosurgeons or orthopedists. The sparsity of material dealing with this topic in the otolaryngologic literature has prompted us to present this paper on the symptomatology, etiology, diagnosis and treatment of this interesting condition which is so prevalent in our specialty. Variations of this symptom complex are referred to in the literature as the cervical syndrome, the cervical root syndrome, the radicular syndrome, and the root compression syndrome. We have elected to designate the condition as the cervico-occipital syndrome because the term best describes the area with which we are concerned.

The initiating cause or causes of this symptom complex can vary. The clinician is often faced with a totality of reactions; therefore, closely related etiologic factors cannot always be separated. The commonest cause, however, is mechanical compression or irritation of one or more cervical nerve roots or portions thereof. Davis[10] in a comprehensive treatise on the subject of radicular syndromes lists a variety of causes, such as osteoarthritis of the cervical spine and trauma. Whip lash injuries, wrenching or straining, blows to the head in a fall, jarring of the spine as in sports, or a number of major or minor accidents can be sustained by the patient, resulting in damage of the intervertebral discs or severe trauma to the nerve roots. The symptoms may come on immediately or a latent period may elapse, depending on the extent of the injury and the development of changes initiated by the original injury. Days and even years may elapse before symptoms appear. Postaural strain may place tension on the spinal ligaments and cervical musculature, eventually resulting in cervical root compressions. Protrusions or herniation of cervical discs is listed as a common cause of symptoms, but Jackson[24] doubts that disc disturbances *per se* are likely to cause compression. According to the latter author, 90 per cent of the patients gave a history of recent or remote injury. Root sleeve fibrosis with or without disc involvement or osteoarthritis can cause radicular changes. Emotional stress causing muscle contractions in the neck, shoulders or back can be a factor in the causation of symptoms. Judovich[27] advances the interesting theory that skeletal muscle spasm of the cervical spine and scalp associated with tension headaches may be due to an intrinsic muscle disturbance related to the release of a stress substance in

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the hypothalamus which triggers the pain mechanism. Finally, any disease process, from an inflammatory reaction to a neoplasm, may involve the cervical nerve roots and cause symptoms in the head and neck.

The concept of myofascial genesis of pain as proposed by Travell and associates[38,39] is worthy of mention as a possible cause of the symptom complex under discussion. Although primarily interested in the role played by the voluntary muscles in pain syndromes of the chest and arm, much of what they say can be applied to the role of voluntary muscles in the head and neck. According to their concept, the trigger area is a small region from which impulses bombard the central nervous system and give rise to referred pain, which is set off whenever the trigger area is stimulated by thermal influences, pressure or needling; or by motion which stretches the structures containing the area. Resistance to stretching may result in shortening of the affected muscle and limitation of motion and weakness. The constant pattern of pain distribution in different individuals indicates that the impulses of somatic pain follow fixed anatomic pathways. This fact enables one to use a known reference pattern to locate the myofascial source of pain. The myofascial trigger mechanism may be initiated by direct trauma to muscle or joint, acute myositis, arthritis, hysteria or nerve root injury. Myofascial pain following stimulation of the trigger area is thought to depend on a reflex pattern maintained by the trigger area. Local block procedures such as procaine injection or spraying with ethyl chloride often terminates the pain cycle. This fact implies that the mechanism which sets the pain cycle in motion is not the same as that which keeps it going. The pain cycle may continue long after the precipitating cause has vanished.

SYMPTOMS

The otolaryngologist sees patients with this affliction when the head and neck manifestations of this syndrome predominate. He is not likely to be consulted because of pain, numbness or tingling in the arms and hands, or pains in the chest, back or shoulders. The bronchoesophagologist might see these patients because of respiratory distress, which occasionally may be the major or even the sole manifestation of nerve root compression. The breathing may be difficult or forced, and at times the patient may complain of inability to inspire or expire air. Coronary artery disease may be simulated by radicular irritation, but the patient does not feel chest compression as he does in angina and the respiratory distress occurs usually at rest with or without chest pain. Although this paper does not concern itself with lower cervical and upper thoracic root compression syndromes, mention is made of them because the otolaryngologist should carefully question the patient as to the presence or absence of such symptoms and their extent. Such inquiries are in order, for they aid in establishing the diagnosis and determining the extent or severity of the root compression.

The head and neck manifestations of the cervico-occipital syndrome are of primary concern to the otolaryngologist. These include headaches in the occipital, parietal, frontal and temporal regions; head pains suggestive of sinus disease, ocular pains and transient blurring of vision; otologic symptoms, such as vertigo, tinnitus, transient hearing loss, sensation of fullness in the ear and pain in and about the ear suggesting mastoid disease or involvement of the middle and external ear; pains about the temporomandibular joint suggestive of the mandibular joint syndrome of Costen; throat disturbances; and finally facial pains suggestive of tic douloureux. We

made a study of these manifestations of the cervico-occipital syndrome in a series of patients. Our findings and impressions constitute a portion of this presentation.

The complaint of headache and pain in the head were the most frequent symptoms encountered in our series of cases. The headaches usually centered in the occipital region with radiation to the frontal, parietal and temporal areas. The patients described the pain as being superficial rather than deep, using such descriptive terms as "my scalp aches" or "my head burns." Some obtained relief by pressing on the head with their hands. Specific areas of localized pain were not uncommon. One patient would knot a scarf to correspond with the areas affected and lessened the pain by tightening the scarf in tourniquet fashion about the head.

The pain at times was dull, throbbing and boring in character, while at other times it was sharp, piercing and lancinating. Frequently the areas involved would remain tender and "sore" between attacks. The pain could be increased or relieved by certain head and neck positions or movements. Recumbency tended to relieve the symptoms or at times made them worse. Some patients were awakened at night with the pain and could obtain relief by changing positions in bed. Sneezing, coughing, or straining often aggravated the pain. If the patient awoke with the head pain, it tended to disappear after the patient was up and about. Changing from a certain position which had been maintained for a time would aggravate the symptoms. Forceful flexion or extension of the head was avoided, for such movements could induce the headaches. Some patients noted an audible crepitation of the neck, particularly on rotation.

Although unilateral in the majority of cases, a bilateral spread of the head pain was not uncommon. We observed that in such instances the pain and discomfort was more marked on one side of the head. In almost all of the cases there was an associated aching in the posterior cervical region and tenderness could be elicited by pressure applied to the region of the second cervical vertebral body. The migraine type of headache was encountered, but it responded well to treatment of the spine. Overton[32] suggests that some irritation of the carotid sympathetic plexus may be involved. Some of our patients complained of blurred vision with the headaches.

Jackson,[23] in reviewing two hundred cases of cervical syndrome, found headache as one of the principal symptoms in one hundred patients. Twenty per cent complained of blurred vision. This finding led her to believe that a sympathetic factor was involved. According to the author, spasm of the neck muscles could produce an irritation of the ganglion or the postganglionic fibers resulting in eye changes such as blurring of vision. Various trigger mechanisms would initiate or aggravate the symptoms, such as exposure to drafts, emotional stress or excessive muscle fatigue.

We noted a marked variance in the degree and extent of the head pain and its distribution. As in all cervical root involvement, this is primarily due to the number of fibers compressed and degree of damage sustained by them. However, as Wolff[40] points out, the individual's capacity to tolerate pain or react to it is not directly proportional to the amount of pain perceived. The emotional stability of the particular patient concerned had much to do with the manner and degree of reaction to the subjective feeling of pain in his head.

Early in our studies of the cervico-occipital syndrome, we were attracted by the work of Costen[2,4] on the mandibular joint syndrome because of the similari-

ties between the two symptom complexes. The parallel findings are readily understandable when one considers that the trigeminal nerve is often involved in the production of symptoms in both instances. Costen[3] found that headaches and ear symptoms predominated in his series of cases. Ear symptoms were next in frequency of occurrence to head pains in our series. Otalgia was the most prominent feature. In most instances we could find no organic cause for the patient's complaint of earache. The pain was localized deep in the ear, below the ear in the region of the mastoid tip and styloid process, and occasionally anterior to the tragus or over the mastoid. In questionable cases, when roentgenographic studies were made of the mastoid, they were found to be normal. At times the patient complained of deafness but, like Costen,[7] we found that this was a subjective reaction of the patient which was not substantiated by audiometric tests. Tinnitus occurred in a number of cases but was not a disturbing feature to the patient. It was usually low pitched and roaring in type and tended to disappear as the patient's general symptoms subsided under treatment.

Upper cervical root compression was considered by Davis[9] as being a common cause of true objective vertigo. He based his concept of the mechanism of cervical vertigo on Spector's analysis of the neuroanatomical systems underlying vertigo.[37] The correlation of impulses coming from the eye, ear and proprioceptive organs of the neck, trunk and extremities serve to orientate one's body in space. The medial longitudinal bundle extends into the cord and coordinates the vestibular apparatus with the eye and anterior horn cells of the spinal cord. This primary coordinating system could be so disturbed by a bombardment of afferent impulses caused by nerve root pressure that vertigo would result. Davis noted that nystagmus, usually present when there is intracranial irritation of the medial longitudinal bundle, was not observed in his patients. Vertigo of varying degree was often associated with occipital headaches in some of our patients. A number complained of a "dizzy headache" or "sick feeling," while others felt unsteady when turning their heads or sitting up from a reclining position. Nausea and vomiting occurred in some instances. These symptoms tended to subside as the patient improved under treatment.

Not infrequently our patients would complain of pain in or about the mandibular joint with radiation down the ramus, but examination failed to reveal tenderness over the joint. Jaw movements had little effect on the degree of pain, and other criteria of mandibular joint involvement[6] were lacking.

Some patients complained of facial pains simulating tic douloureux, but the symptoms were milder, the radiation of pain was atypical and a trigger mechanism was lacking or inconsistent. It is interesting to note that Gardner and Miklos[13] feel that trigeminal neuralgia may be localized to the posterior root of the fifth cranial nerve just as sensory disturbances can be localized to the posterior root of spinal nerves as in brachialgia and sciatica.

Other symptoms, such as vague throat pains usually reported deep and throbbing in character, glossodynia, dysphagia and disturbances in taste, were noted, but their occurrence was too infrequent to warrant any deductions as to their significance. They are worthy of mention, for they suggest the possible involvement of the seventh, ninth and tenth cranial nuclei in the symptom complex.

PATHOGENESIS OF SYMPTOMS

The term cervico-occipital syndrome does not imply a diagnosis. It is a descriptive term applied to a symptom complex resulting from various and often obscure disease processes. A discussion of the pathogenesis of these symptoms is warranted, for it sheds a light on the disease process and the manner in which the pain and discomfort suffered by the patient is produced. Such knowledge is essential before a diagnosis can be made and treatment attempted.

A review of the anatomy peculiar to the cervical vertebrae helps to clarify some of the problems associated with the syndrome.[30] Of the seven cervical vertebrae, the first two, the atlas and axis, differ from the remaining five. The motion between the atlas and skull is minimal. Motion between the atlas and axis consists of nodding and rotation of the head with lateral bending. Motion of the remaining cervical spine is essentially forward flexion and extension with slight degree of rotation.

Hunter and Mayfield[22] stress the role of the upper cervical roots in the production of pain in the head. The first cervical nerve root emerges between the occiput and the atlas (C-1). Since there is little motion between the atlas and the occipital bone and since the sensory component of C-1 is rudimentary and even at times absent, this root seldom plays a part in the production of symptoms. The situation regarding the axis (C-2) is vastly different. The joint between the atlas and axis is highly movable, placing unusual stress on the anterior primary ramus of C-2. Furthermore, the posterior primary ramus of C-2 is not protected posteriorly by pedicles and facets which complete the root canal elsewhere in the vertebral column but lies exposed on the posterior aspect of the vertebra. The third cervical nerve is well protected within its bony canal and is no more vulnerable to injury than the remaining cervical nerve roots. The posterior primary ramus of C-2 becomes the greater occipital nerve and supplies sensation to the major portion of the scalp and overlaps into the face areas. It seldom ends at the vertex but usually goes as far as the forehead.[41] The relatively large number of patients whom we were able to relieve by infiltrating local anesthetic agents into the area of the greater occipital nerve in the posterior aspect of the neck leads us to believe that irritation of this nerve root (C-2) played a primary role in the production of head symptoms in the majority of cases in our series.

The remaining five cervical nerves emerge through intervertebral foramina, which are like canals with long vertical diameters and short anteroposterior diameters. The posterior walls are formed by the articular processes of the joints, while the superior, anterior and inferior walls are formed by the pedicles and posteriolateral portions of the bodies. The cervical nerve roots as they course through these canals are vulnerable to hemorrhage, inflammatory processes of the capsule, spurs, pressure or irritation from ruptured discs and abnormal motion of the joints due to relaxation or tearing of the capsular and ligamentous structures.

It is generally believed that the symptoms associated with the cervical root syndrome are caused by a reflex mechanism due to irritation of the cervical nerve roots producing pain anywhere along the segmental distribution of the nerve. The multiplicity and complexity of the head and neck manifestations of the cervico-occipital syndrome force the otolaryngologist to seek a more adequate explanation of the pathogenesis of these symptoms. Although some authors[34] feel that a satisfactory explanation of the mechanism by which a pathologic cer-

vical disc can produce headaches does not necessarily include evidence of direct neural connections between the site of the pathologic disc and the region in which the patient believes the headache to be located, we are of the opinion that neural connections do exist that aid in explaining the mechanism by which such symptoms are produced. Such an explanation must be based on the fact that the cranial nerves are involved in the production of the symptom complex. We can better understand our basic problem if we accept De Jong's concept that the individual cranial nerves, especially the last ten pairs, are similar in structure and function to the spinal nerves and react similarly to disease processes.[11] The first two cranial nerves and, to a certain extent, the eighth nerve react more as does the brain proper. He states further that owing to the proximity of many of the cranial nerve nuclei, especially those in the brain stem, to other structures, it is rare to have a single nucleus affected without other involvement. This is well demonstrated in cranial nerve involvement in poliomyelitis.[12] Mention has already been made of Gardner and Miklos' comparison of the fifth cranial nerve to a spinal nerve.[13]

In what manner could the cranial nerves become involved in the production of the symptoms under discussion? Josey[26] in attempting to explain the cause of headaches associated with pathologic changes in the cervical part of the spine states that it is well known that the sensory nucleus of the fifth cranial nerve extends down the spinal cord to the level of the third cervical segment but there are no known synapses with the upper cervical sensory nerves. He admits, however, that such a possibility does exist. Campbell and Parsons[1] mention that there is a profuse interconnection between the cervical plexus and cranial nerves VII, VIII, IX, X, XI, and XII, and the superior sympathetic ganglia, but they do not elaborate upon this statement.

Since the fifth cranial nerve is primarily involved in the symptom complex under study, a brief review of its anatomy and function is in order. The cells of origin of the sensory portion of the trigeminal nerve have their nuclei in the gasserian ganglion. The neuraxes from the gasserian ganglion terminate in two sensory nuclei. The chief sensory nucleus, also known as the pontine nucleus, is situated in the pons and receives only tactile stimuli. From here the neuraxes, carrying discriminating and tactile sensations, ascend to the thalamus. The second sensory component is known as the descending root or spinal tract of the trigeminal nerve and extends downward from the chief sensory nucleus through the lower pons and medulla into the upper portion of the spinal cord. Humphrey[18] has demonstrated that in the human embryo the spinal tract of the fifth nerve may even extend to C-4. Pain, temperature and general tactile impulses terminate on this tract and nucleus. The fibers descend for various distances, then synapse, and neuraxes and neurones of the second order cross and ascend to the thalamus in the secondary ascending tract of the trigeminal nerve. The external branches of the gasserian ganglion pass outward as the sensory root (posterior root) of the trigeminal nerve, which then divides into the ophthalmic, maxillary and mandibular divisions.

Sjöquist[35] showed by fiber analysis that the majority of the touch fibers remain in the cephalic portion of the sensory nucleus of the fifth nerve and that the pain and thermal fibers descend down its spinal tract. This finding gave him the idea for a new operation to relieve facial pain of trigeminal origin by sectioning the spinal tract of the fifth nerve at the lower brain stem level. He applied the term *trigeminal tractotomy* to this

operation. Sjöquist also pointed out that the proximity of the sensory nucleus of the seventh nerve to the spinal nucleus of the fifth nerve might play a role in the origin of facial neuralgia which persists after sectioning of the fifth nerve tract. Grant and his associates[14,15] made further observations on the results of the trigeminal tractotomies which aid in understanding the function of the descending root of the fifth cranial nerve.

Phylogenetic studies and basic studies on human embryology by neuroanatomists likewise aid in explaining the probable mechanism involved in the production of symptoms associated with the cervico-occipital syndrome. Crosby and Yoss[8] point out that phylogenetic history frequently sheds light on some of the factors determining the sequence of embryologic development of brain stem centers and tracts and on probable functional significance of the adult areas. They have shown that a cutaneous sensory component occurs in the seventh, ninth and tenth cranial nerves in many vertebrates including man. The sensory roots of the ninth and tenth nerves usually cross the spinal tract of the fifth nerve and contribute fibers to it as they pass. Within the tract they descend to terminate in the subnucleus caudalis of the spinal tract of the fifth nerve. The best evidence for this termination in man is the insensibility of the ear to painful stimuli following trigeminal tractotomy at the lowermost brain stem levels. In a study of the comparative anatomy of the nervous system of vertebrates, Kappers and his associates[28] point to the close association of the descending course of the trigeminal nerve and the ascending course of the spinal nerves as an illustration of the theory of neurobiotaxis, for these courses indicate the tendency of centers which frequently receive simultaneous or successive stimuli to approach each other. Correlative function is the fundamental fact determining the arrangement of neural cells and their dendrites. Larsell and Fenton,[31] in discussing basic concepts of the cause of otalgia, likewise express the opinion that components of similar functional type tend to group together.

Hooker[16] in his studies on fetal behavior states that in the development of human fetal activities the cutaneous areas supplied by the trigeminal nerve mediate all of the earliest responses to exteroceptive stimulation. The first sensory area is restricted to the lips and nasal alae. The usual response to unilateral trigeminal stimulation is movement of the face away from the stimulus in avoiding reaction. The human embryo first responds to exteroceptive stimulation at seven and one-half weeks of menstrual age following stimulation of the nose-mouth area. The resulting reflex consists of a contralateral axial flexion limited to the cervical region. This reaction obviously involves the maxillary fibers of the trigeminal nerve and the ventral horn motor neurones in the cervical region of the spinal cord. Humphrey[20] in discussing the trigeminal nerve in relation to early human fetal activity states that the cutaneous sensory fibers of the seventh, ninth and tenth nerves pass through the spinal tract of the fifth nerve in the early embryo and retain essentially the same pattern in the adult. On the basis of studies carried out on ten human embryos between seven and one-half and eight and one-half weeks of menstrual age, Humphrey[19] was able to show that all three divisions of the fifth nerve pass as far caudally as C-2, and some even reach C-4. The bulk of the fibers ended in the upper half of C-2. These findings corroborate the work of Sjöquist mentioned earlier in this paper. In commenting upon the termination of the fibers of the trigeminal nerve in relation to the gray matter

of the upper cervical cord, Humphrey[21] emphasizes three points: (1) the existence, in these upper cervical cord levels, of a sensory fiber pattern for the entire embryo; (2) the appearance of a primitive center of centralization or integration in the upper cervical region of the spinal cord, and (3) the presence of the most caudal part of the nucleus of the spinal tract of the trigeminal nerve in the upper cervical spinal cord levels, where it is included customarily as part of the dorsal horn gray matter. These basic observations on early fetal development and behavior are important in the understanding of our problem relating to the close association of the cranial nerves, particularly the trigeminal nerve, with the upper cervical spine. Hooker[17] states that during the earlier part of fetal life, the behavior that develops is purely reflex in nature. The intricate nervous mechanism which makes possible the continuing patterns of reflex behavior lay the foundation for future voluntary acts by the postnatal child. The receptors, the internuncial neurones and the neuromuscular connections used in early fetal reflex behavior are the structural basis on which later action patterns are built. This is retained and utilized in the development of postnatal behavior. Not even the reflex arcs are discarded as such. They continue to serve the organism throughout its life.

From the evidence herein presented, it is logical to assume that the head and neck manifestations of the cervico-occipital syndrome are primarily due to the close relationship of the cervical spinal nerves and the cranial nerves. The spinal tract of the trigeminal nerve and its sensory nuclei are the prime mediators by which this relationship is established. The close association of the sensory nuclei of other cranial nerves, particularly those of the seventh, ninth and tenth nerves, with those of the fifth cranial nerve provide pathways for communication between these centers in the brain stem and centers in the cervical spine. The similarity in structure of the cranial nerves to spinal nerves implies similarity in function. It is understandable, therefore, that afferent impulses originating in the cervical region from irritation of the cervical nerve roots can initiate responses that are mediated *via* various pathways in the head and neck. The otolaryngologist needs no convincing that the converse can also happen, for he constantly observes patients with pain in the cervical area from pathologic disturbances in the nose, sinuses, ear, tongue, pharynx and larynx.

DIAGNOSIS

The diagnosis of a cervical nerve root disorder is made on the history, symptoms, clinical findings and roentgenographic studies of the cervical spine. Response to treatment helps to confirm the diagnosis. Objective neurologic changes are rare, except in cases of long standing or when damage to the nerve root is unusually severe.

In a majority of cases the patients give a history of antecedent trauma to the head and neck. The injury may have been of such minor degree that the patient was unaware of its significance. In some instances a history of previous trauma may be entirely lacking. There may be periods when the patient may be free of symptoms, only to have them recur following exposure to physical or emotional stress.

The symptoms associated with the head and neck manifestations of the cervico-occipital syndrome have already been enumerated. Their severity and extent depend upon the particular nerve root or roots involved and the severity of the injury sustained by the nerve. Motions of the head and neck produce or aggravate the pain. Suboccipital head-

aches can at times be relieved by certain head positions, especially hypertension. Vertigo can be produced by changing the head from a fixed position. Recumbency, especially during rest in bed is often related to the occurrence of symptoms because of alteration in the cervical spine. Fixed postures such as may be assumed at work can cause symptoms toward the end of the day. Paresthesias of various kinds may affect the head and neck region and may be a prominent part of the symptomatology. Keegan[29] stresses that hypalgesia can occur with the loss of a simple nerve root. Limitation of motion of the neck, tightness or knots in the muscles of the neck and pains in the supraclavicular region may be complained of.

The clinical findings are related to the nerve roots involved. Tenderness to deep pressure over the spine is almost invariably present, especially on the side of nerve root involvement. Even mild pressure may cause acute pain. We have found tenderness over the area of exit of the greater occipital nerve in almost all of our cases. The mastoid region may also be tender. Not infrequently, tenderness could be elicited by pressure over the temporal region or the frontal sinus area. In the latter instance there was no clinical evidence of frontal sinus disease. Relaxation of the cervical spine is important when examining the neck. If the neck is slightly extended, the upper cervical spinous processes can be more readily palpated. The transverse processes can be palpated from just below the mastoid process to the angle of the neck and shoulder. The head should be held in various positions when it is being examined for tenderness. If the patient states that certain specific body movements induce pain, an attempt should be made to reproduce the pain by having the patient assume these positions. Neck compression may reproduce the pain when the head and neck are tilted towards the affected side and pressure applied over the top of the head. Pain produced when the patient is asked to cough or strain may be a diagnostic sign. We have found that Travell trigger areas, mentioned earlier in this paper, played a prominent part in the symptomatology and could frequently be elicited upon palpation. The application of traction is an aid to diagnosis and a guide to therapy. As a rule traction relieves the patient's symptoms, but at times it may induce symptoms or make them worse. Manual traction performed at the time of examination while the patient is lying supine may be of diagnostic importance.

Roentgenographic studies are essential. Three lateral views are made with the tube at a distance of seventy-two inches. One view is taken with the patient looking straight ahead, the other with the neck in flexion, and the third with the neck in hyperextension. The straight lateral view reveals compression, marginal lipping, narrowed intervertebral spaces, fusion of vertebrae, and other anomalies. Obliteration or diminution of the normal lordatic curvature of the spine due to spasm of the neck muscles is very often demonstrated. The chin or chest view reveals forward subluxation, while hyperextension reveals posterior subluxations. Oblique views are taken to show the intervertebral foramina and may show spur formation. The anteroposterior view may reveal fractures, hyperplastic changes, and other anomalies such as a cervical rib. Myelography may be indicated to show disc protrusions not demonstrated by other techniques.

The differential diagnosis of cervico-occipital syndrome is predicated upon a thorough ear, nose and throat examination. Pathologic disturbances in the head and neck other than nerve root irritation might initiate pain stimuli mediated to

the cervical nerve root distribution via the sensory nuclei and efferent and afferent pathways of the cranial nerves or by direct involvement of the sensory root fibers of the spinal nerves, particularly the greater occipital nerve. The differential diagnosis must also include mandibular joint neuralgias and other cranial neuralgias, which are well discussed by Costen.[5] Referred pain from visceral or somatic structures which have the same segmental nerve root supply may be accompanied by superficial tenderness or hyperalgesia but not by deep tenderness or muscle spasm. Reflexes are usually normal. Pain which results from lesions of the cervical spinal cord and of the brain follows no definite root pattern and is ill defined and indistinctly demarcated. Hypertonia and hyperactive reflexes are the rule in such cases.[25]

TREATMENT

Conservative approach is the rule in treatment. Rarely is surgical intervention necessary. In our series of cases, in only two instances was decompression by surgical means indicated. Treatment should be aimed at correcting or improving the mechanics of the neck. Correction of poor posture may be sufficient to give relief. Rest and avoidance of certain neck and trunk movements may permit soft tissue reactions to subside. Jackson[23] stresses correct sleeping posture and has devised a contour pillow which maintains the natural curve of the neck. Sometimes a Thomas collar or other means of immobilizing the cervical spine may rest the spine when the patient is out of bed.

Heat and other forms of physiotherapy aid materially in reducing symptoms. Hot wet compresses or dry heat over the cervical spine gives relief when symptoms are acute. Radiant heat, infrared radiation or diathermy may also be applied. At times heat may aggravate symptoms and should be discontinued. The heat should be applied for fifteen to twenty minutes about every three or four hours. Gentle massage with the application of a mild lotion liniment is helpful to relax muscle spasm.

Temporary, and at times permanent, relief may be obtained by infiltrating the skin or subcutaneous tissues with a local anesthetic agent. We use 0.5 per cent procaine if a large area is to be infiltrated, and 1.0 per cent Xylocaine for more localized infiltration, as for blocking the greater occipital nerve. Occasionally Travell's technique of using an ethyl chloride spray over the affected area of the neck aids in controlling trigger areas. Pentecost and Adriani[33] found blocking of C-2 and C-3 to be effective in the diagnosis and treatment of cephalalgias of cervical origin. Muscle relaxing drugs and salicylates aid in controlling symptoms. Tranquilizing drugs may be indicated for those patients in whom emotional factors tend to accentuate symptoms. Patients should be cautioned to avoid exposure to drafts and rapid environmental changes, such as stepping into an air-conditioned establishment when the outside atmosphere is hot and humid. We have seen acute attacks precipitated in our own waiting room when patients with this syndrome sit for a period of time near an air-conditioning outlet.

Traction is the most effective method of treatment in most cases. However, not all cases respond to traction and some individuals may even be made worse by this form of treatment. If symptoms are aggravated, traction should be discontinued. Older individuals do not tolerate the procedure as well as does the younger age group. Smith[36] advocates the use of a Sayer halter traction with a "fishscale" attachment, which he feels is more effective than cervical

collars or bed traction. He cautions against the use of traction in cases showing complete bridging between vertebrae in the cervical area. Traction reduces muscle spasm, increases the intervertebral spaces, stretches the ligaments, increases the size of the intervertebral foramina and decreases pressure on the nerve roots. Traction may be continuous or intermittent, with the patient in an upright or a reclining position. The amount of weight applied depends upon the patient's tolerance and the effectiveness of the traction. Shifting of the direction or angle of traction often gives relief when straight upward traction is not effective. Adjusting the head halter is important. As much weight as possible should be put on the occipital strap. In order to relieve pressure on the temporomandibular joint, a rubber mouth guard or disc should be inserted between the teeth. All dentures should be removed. The duration and frequency of treatments depends upon the relief obtained from them. It may be desirable for the patient to purchase a traction apparatus for use at home.

If the patient does not respond to the treatment as outlined, a consultation with a neurosurgeon or orthopedist is indicated. The majority of our patients received adequate or complete relief of symptoms by the conservative form of therapy.

PRESENTATION OF CASES

A study was made of seventy-five patients seen in our practice who had head and neck manifestations of the cervico-occipital syndrome. The ages varied from 9 years to 68 years, with an average age incidence of 41.7 years. The condition predominated in females. In contrast to the findings of other authors, a history of recent or remote antecedent trauma to the head and neck prior to the onset of symptoms was elicited in a relatively low percentage of cases. Forty-one per cent gave a past history of trauma, and 14 per cent gave a history of trauma immediately preceding the onset of symptoms. This may be explained by the fact that the symptoms we observed were mild compared to the disabling conditions seen by orthopedists. The initiating trauma may also have been less severe and consequently may have been overlooked or promptly forgotten by the patient.

The symptoms ranged from mild attacks of pain in the head and neck to severe incapacitating illness which required hospitalization. All the patients

TABLE I

SYMPTOMS IN FORTY-ONE CASES WITH NO EAR DISEASE

SYMPTOM	NO. OF CASES	% (BASED ON 75 CASES)
Pain	34	45.0
Hearing Loss	14	19.0
Vertigo	16	21.0
Fullness	8	10.6
Tinnitus	6	8.2
Nausea and Vomiting	5	6.6

complained of pains in the head and neck. Of forty-nine cases with symptoms referable to the ear, only eight had proven ear disease.

Table I shows the distribution of symptoms among the forty-one patients with no demonstrable ear disease. Pain in and about the ear predominated. The complaint of hearing loss in the fourteen cases listed could not be substantiated by audiometric studies. The vertigo complained of was directly related in degree of severity to the head pain which the patient suffered.

Eleven patients complained of deep throbbing pains in the throat on the affected side. The examination of the throats of these patients failed to show any signs of infection or irritation. Miscellaneous symptoms, such as parasthesias about the head, neck and face (8 cases), ocular symptoms (6 cases), and pains simulating sinus disease (4 cases), were noted. No pathologic findings were evident to explain these symptoms. It was interesting to note that most of the symptoms described subsided or were alleviated entirely as the patients responded to treatment.

On physical examination, all of the patients demonstrated tenderness of varying degree over the distribution of the greater occipital nerve, along the spinous processes of the cervical vertebrae or down the back. Areas of tenderness, as described by Travell,[39] could be elicited in some cases. Two of the cases had proven mandibular joint disturbances in addition to the cervical occipital syndrome. Roentgenograms of the cervical spine were not taken routinely. In the majority of instances in which roentgenograms were taken, these showed changes in the cervical spine which could account for the patient's symptoms. Occasionally the roentgenographic studies were negative even though all of the other findings fulfilled the criteria for a diagnosis of cervico-occipital syndrome.

Treatment was primarily conservative. Surgical intervention was necessary in only two cases. In two other cases, neoplastic disease was found to be the cause of cervical nerve root irritation resulting in symptoms typical of this syndrome. The remaining 71 cases responded well to conservative therapy consisting of infiltration of the nerve root or the greater occipital nerve with local anesthetic agents, use of ethyl chloride sprays, physiotherapy and traction given at the office or self administered at home, and the judicious use of medication such as muscle relaxing agents, tranquilizers, pain relieving drugs, sedatives and lotion liniments. We found that reassurance with consequent relief from anxiety did much to improve the patient's symptoms. They were taught to live with their affliction and to adjust themselves to it. Avoidance of exposure to noxious agents and situations such as drafts, poor posture, stress situations, etc., which bring on an attack was advised. Most of the therapy was carried out at home or in the office. Only twelve patients required hospitalization for more extensive treatment.

A brief résumé of four hospitalized cases is presented. They illustrate the more serious complications occasionally seen in patients with a cervico-occipital syndrome.

Case 1.

E. G., a 28-year-old white woman, was first seen in May of 1948 with the complaint of pain in the right ear. The pains were sharp and stabbing in character and were most severe in the ear canal. She gave no history of antecedent trauma to the head or neck. Findings in a complete ear, nose and throat examination were normal. The patient showed marked tenderness in the neck of the involved side. A diagnosis of cervico-occipital syndrome was made and conservative therapy

instituted. The patient obtained some relief but the pain recurred periodically. In the fall of 1956, she moved to another city. In December of that year she returned for further consultation because she had been told that a mastoidectomy was mandatory to relieve her pain. Careful examination of the ear failed to reveal any indication for the operation. She was referred to a neurosurgeon, who performed a cervical laminectomy, decompressing C-5 and C-6 on the right. The patient has been symptom free since December 1956.

Case 2.

M. F., a 34-year-old white woman, was seen in March 1957. She complained of pains in the right side of her head and neck radiating down the right arm. The symptoms came on following a head injury five years prior to date of examination. She also complained of tinnitus and fullness in the right ear, accompanied by attacks of dizziness. Examination revealed tenderness over the right neck region. The ears appeared to be normal. Roentgenograms of the cervical spine showed a narrowing of the foramina between C-4 and C-5 on the right. Since her symptoms persisted in spite of all forms of conservative therapy, the patient was admitted to the hospital for a decompression operation in June 1957. Operative findings showed marked fibrosis about the root of C-5. The patient's ear symptoms disappeared following the operation and she has been symptom free to date (October 1959).

Case 3.

J. N., a 36-year-old white woman, was seen in September 1959. She gave a history of pain which began in the right side of her neck, then gradually spread to the right side of the head, the right eye and cheek area and right side of the nose. Occasional pains were felt in the right ear. Physical examination revealed tenderness over the right neck and occipital region. The right eye and ear were found to be normal. A diagnosis of cervico-occipital syndrome was made and the patient was placed in traction. Since her symptoms were not relieved, she was admitted to the hospital for further studies.

Roentgenograms of the base of the skull revealed an area of decreased density in the region of the foramen rotunda and the foramen lacerum. Multiple biopsies of the nose and nasopharynx failed to reveal any tumor. A diagnosis of a neoplasm involving the base of the skull was made and an explanatory craniotomy advised, but the patient refused the operation. When last seen in October 1959 her symptoms showed progression the pain was more widespread and more severe.

Case 4.

M. M., a 79-year-old white woman, the mother of a physician, began to complain of vague neck pains on the left side early in November 1958. The pains gradually spread until they involved the left side of the head and neck and radiated down the left arm. She also complained of a persistent sore throat, but findings of the ear, nose and throat examination were normal. A diagnosis of cervico-occipital syndrome was made and conservative therapy instituted. Since no relief was obtained by these measures, consultants were called to confirm the diagnosis. They felt that the symptoms were caused by osteoarthritic changes in the cervical spine. The symptoms continued to progress in spite of intensive treatment. She was admitted to the hospital for further studies in May 1959. Neurological examination showed marked weakness in the extremities with increased reflex activity. The spinal fluid sugar was found to be 38 mg. and the total protein 243 mg. Roentgenograms of the cervical spine showed extensive osteolytic deterioration of the fourth cervical vertebra and partial destruction of C-3. Further clinical and laboratory studies failed to show any primary tumor. An exploratory operation of the cervical spine revealed destruction of C-4. The biopsy specimen showed undifferentiated adenocarcinoma. The patient died on the twelfth postoperative day. Autopsy revealed a solitary alveolar cell carcinoma in the right upper lobe of the lung with metastasis to the cervical vertebra.

Cases 1 and 2 illustrate ear symptoms frequently seen in patients with cervico-occipital syndrome. Cases 3 and 4 illustrate how a relatively benign symptom complex such as the cervico-occipital syndrome can portend serious and even fatal disease.

SUMMARY

A symptom complex designated as the cervico-occipital syndrome has been described. Symptomatology, etiology, diagnosis and treatment were discussed. Evidence was presented to support the assumption that head and neck manifestations of the cervico-occipital syn-

drome were due primarily to the close relationship of the sensory components of the spinal nerves and the cranial nerves. Irritation of the cervical nerve roots results in reflex pain patterns mediated by these centers and their efferent pathways. The importance of the spinal tract of the trigeminal nerve in the production of symptoms was stressed. Case reports were presented.

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REFERENCES

1. Campbell, Douglas G., and Parsons, Clare M.: Referred head pain and its concomitants. Report of preliminary experimental investigation with implications for the post-traumatic "head" syndrome, J. Nerv. & Ment. Dis., 99:544-551 (May) 1944.

2. Costen, James B.: A syndrome of ear and sinus symptoms dependent upon disturbed function of the tempomandibular joint, Ann. Otol. Rhin. & Laryng., 43:1-15 (March) 1934.

3. ———: Neuralgias and ear symptoms, associated with disturbed function of the temporomandibular joint, J.A.M.A., 107: 252-255 (July 25) 1936.

4. ———: Reflex effects produced by abnormal movement of the lower jaw, Arch. Otolaryng., 36:548-555 (Oct.) 1942.

5. ———: Diagnosis of mandibular joint neuralgia and its place in general head pain, Ann. Otol. Rhin. & Laryng., 53: 655-659 (Dec.) 1944.

6. ———: Differential diagnosis of mandibular joint neuralgia, Clin. Med., 4:1095-1099 (Sept.) 1957.

7. ———: Outline of the mandibular joint syndrome, Laryngoscope, 69:408-414 (April) 1959.

8. Crosby, Elizabeth C., and Yoss, Robert E.: The Phylogenetic Continuity of Neural Mechanisms as Illustrated by the Spinal Tract of V and Its Nucleus. Chapter XII in Genetics and the Inheritance of Integrated Neurological and Psychiatric Patterns (Research Publications of the Association for Research in Nervous and Mental Disease), Baltimore, Williams & Wilkins, 1954, vol. 33, pp. 174-208.

9. Davis, David: A common type of vertigo relieved by traction of the cervical spine, Ann. Int. Med., 38:778-786 (April-June) 1953.

10. ———: Radicular Syndromes, with Emphasis on Chest Pain Simulating Coronary Disease, Chicago, The Year Book Publishers, 1957.

11. De Jong, Russell Nelson: The Neurologic Examination; Incorporating the Fundamentals of Neuroanatomy and Neurophysiology, ed. 2, New York, Hoeber-Harper, 1958.

12. Engler, Clarence W., and Missal, Sylvester C.: Involvement of the cranial nerves in poliomyelitis, Tr. Am. Acad. Ophth. & Otol., 59:732-739 (Nov.-Dec.) 1955.

13. Gardner, W. James, and Miklos, Michael V.: Response of trigeminal neuralgia to "decompression" of sensory root; discussion of cause of trigeminal neuralgia, J.A.M.A., 170:1773-1776 (Aug. 8) 1959.

14. Grant, Francis C.; Groff, R. A., and Lewy, F. H.: Section of the descending spinal root of the fifth cranial nerve, Arch. Neurol. & Psychiat., 43:498-509 (March) 1941.

15. Grant, Francis C., and Weinberger, Lawrence M.: Experiences with intramedullary tractotomy. I. Relief of facial pain and summary of operative results, Arch. Surg., 42:681-692, 1941.

16. Hooker, Davenport: Early Human Fetal Behaviour with a Preliminary Note on Double Simultaneous Fetal Stimulation. Chapter VIII in Genetics and the Inheritance of Integrated Neurological and Psychiatric Patterns (Research Publications of the Association for Research in Nervous and Mental Disease), Baltimore, Williams & Wilkins, 1954, vol. 33, pp. 98-113.

17. ———: Evidence of prenatal function of the central nervous system in man. James

Arthur lecture on the evaluation of the human brain, New York, The American Museum of Natural History, 1957.

18. Humphrey, Tryphena: The caudal extent of the descending root of the trigeminal nerve during the period of early human fetal activity, Anat. Rec., 109, 46:47, 1951.

19. ———: The spinal tract of the trigeminal nerve in human embryos between 7½ and 8½ weeks of menstrual age and its relation to early fetal behavior, J. Comp. Neurol., 97:143-209 (Aug.) 1952.

20. ———: The Trigeminal Nerve in Relation to Early Human Fetal Activity. Chapter X in Genetics and the Inheritance of Integrated Neurological and Psychiatric Patterns (Research Publications of the Association for Research in Nervous and Mental Disease), Baltimore, Williams & Wilkins, 1954, vol. 33, pp. 127-154.

21. ———: Pattern formed at upper cervical spinal cord levels by sensory fibers of the spinal and cranial nerves, A.M.A. Arch. Neurol. & Psychiat., 73:36-46 (Jan.) 1955.

22. Hunter, Curwood R., and Mayfield, Frank H.: Role of upper cervical roots in the production of pain in the head, Am. J. Surg., 78:743-751 (Nov.) 1949.

23. Jackson, Ruth: The cervical syndrome as a cause of migraine, J. Am. M. Women's A., 2:529-534 (Dec.) 1947.

24. ———: Cervical syndrome, Am. Acad. Orthopaedic Surgeons, 10:65-90, 1953.

25. ———: The cervical syndrome, Clinical Orthopedics, 5:138-148, 1955.

26. Josey, A. Izard: Headache associated with pathologic changes in cervical part of spine, J.A.M.A., 140:944-949 (July 16) 1949.

27. Judovich, Bernard D.: Muscle tension headache—A new concept, Pennsylvania M. J., 59:901-904 (Aug.) 1956.

28. Kappers, C. V.; Huber, G. C., and Crosby, E. C.: The comparative anatomy of the nervous system of vertebrates including man, New York, MacMillan, 1936.

29. Keegan, Jay: Relations of nerve roots to abnormalities of lumbar and cervical portions of the spine, Arch. Surg., 55: 246-270 (July-Dec.) 1947.

30. Kroll, Harry G.: The cervical syndrome; an irritative process of cervical nerve roots with many and varied symptoms, J. Kansas M. Soc., 59:385-388 (Sept.) 1958.

31. Larsell, O., and Fenton, Ralph A.: The embryology and neurohistology of sphenopalatine ganglion connections; a contribution to the study of otalgia, Laryngoscope, 38:371-389 (June) 1928.

32. Overton, Lewis M.: Cervical spine nerve root pain, Am. Surg., 17:343-350 (April) 1951.

33. Pentecost, Paul S., and Adriani, John: The use of cervical plexus block in the diagnosis and management of atypical cephalalgia of cervical origin, Anesthesiology, 16:726-732 (Sept.) 1955.

34. Raney, Aidan A., and Raney, Rupert B.: Headache: A common symptom of cervical disk lesions, Arch. Neurol. & Psychiat., 59:603-621, 1948.

35. Sjöquist, Olof: Studies on pain conduction in the trigeminal nerve. A contribution to the surgical treatment of facial pain, Acta Psychiat. et neurol., Suppl., 17:1-139, 1938.

36. Smith, R.: The cervical root syndrome, Bull. Benjamin Franklin Clinic, (July) 1956.

37. Spector, Benjamin: Neuroanatomic mechanisms underlying vertigo and nausea, Bull. New England M. Center, 10:145-154 (Aug.) 1948.

38. Travell, Janet, and Rinzler, Seymour: Pain syndromes of the chest muscles: Resemblance to effort angina and myocardial infarction, and relief by local block, Canad. M. A. J., 59:333-338 (Oct.) 1948.

39. ———: The myofascial genesis of pain, Postgraduate Medicine, 11:425-434 (May) 1942.

40. Wolff, Harold George, and Wolff, Stewart: Pain (American Lecture Series, ed. 5). Springfield, Ill., Charles C Thomas, 1951.

41. Zander, Richard: Beitrage zur kenntnis der Hautnerven des Kopfes [Contribution to knowledge of skin nerves of the head], Anat. Hefte, 9:1-77 (Jan.) 1897.

DISCUSSION

JAMES B. COSTEN, M.D., St. Louis: A treatise on the sources of pain in the cervico-occipital area is timely, and Dr. Missal's paper should reactivate the interest of otolaryngologists in an area which produced vast contributions in the decade 1930-1940. Before that time, fibrositis of nuchal muscles was ascribed as a common cause of occipital, tender points being observed along the attachments.

Halle (Monatschr. Ohrenh., 45:768, 1911), Mithoefer (Tr. Am. Laryng. Rhin. & Otol. Soc., 40:284, 1934), and Seydell in 1940 (Arch. Otolaryng., 32:860, 1940) first described indurative and nodular myalgias of neck muscles with headache, the tender nodular areas discovered only by palpation. Reports in otolaryngic literature are scant since then, but so voluminous in general medical and surgical writings as to defy any attempt to review them all. Seydell emphasized the presence of frontal, periorbital and maxillary pain, as well as nausea and vertigo, in connection with painful spasticity of the cervical muscles. In various reports since then Travell (New York State J. Med. 55:1955) reported headache and dizziness associated with a trigger area in the sternomastoid muscle.

Albee (Am. J. Surg., 23:1934) gave the name myofascitis to low-grade inflammation of muscles and fascia, especially marked at fascial insertions of muscle to bone. He considered toxic foci as the source, the main emphasis being placed upon intestinal sources.

"The Cervical Syndrome" was described by Jackson, (Thomas, 1956) as an orthopedic problem of the cervical spine, with occipital, vertex and frontal headache.

Kelly (Med. J. Australia 2:1942) presented cases which seemed to show that lesions situated deeply in the suboccipital region often caused no local pain whatever, being referred wholly to frontal or orbital areas. He indicated that the lesion in many cases of post-traumatic headache was situated in the cervical muscles rather than in intracranial structures. He suggested that cervical muscles may play an important part in production of headaches of truly intracranial origin.

Identity of mandibular joint symptoms are not difficult to separate from those which are cervico-occipital. Both have the common quality of simulating, but not being related to, sinus disease. A small fraction of mandibular joint cases are preceded by injury, a majority of cervico-occipital are so induced.

In the mandibular joint symptom-complex, pain and trismus reflexes are produced by impingement of skeletal upon sensory soft tissues. The trigger is within or about the joint capsule, except when initiated by inflammation of the parotid, masticator muscles, or the external ear canal. We have numerous cases of intractable pain relieved by removing an adherent calcified meniscus, or polishing away exotoses from edges of the condyle. Similarly, Dr. Missal ascribes cervico-occipital pain to irritation of cervical nerve roots by foraminal spurs, fractured articular processes, or displaced cervical discs. In both mandibular joint and cervical cases, the exact area of pain production can be determined with certainty by infiltration with procaine or Xylocaine.

A case in which the mandibular joint syndrome and cervical nerve root irritation occurred in the same patient was seen recently in consultation with Dr. A. J. Cone:

Case 1.

Mrs. J. W., female, age 46, examined on September 24, 1959, described otalgia and vertex pain, on the right side, increased on chewing movement. This had been slightly improved by restoration of occlusion. Further, she had a constant occipital and shoulder pain on the right, which extended down the right arm. No relief could be obtained from any change in position when up or lying down.

Her past history revealed occlusal problems all her life, with many changes. Films of the mandibular joints showed narrowing of joint spaces on the right. Internal palpation showed painful tenderness of the right joint. Nine months ago she sustained a shocking blow to the top of her head, as she raised up beneath a wall cabinet with a heavy package in her arms. The "drawing or burning" soreness from the right occiput downward into the scapular area began at once, increased severely the past month. Films showed narrowed disc space between C_2 and C_3. Palpation located a spot of deep tenderness about the center of the upper border of the trapezius muscle, 8 cm. mesial to the acromial process.

One half a cubic centimeter of Depo-Medrol was injected into the right mandibular joint space, after infiltrating the skin and joint capsule with 1 per cent procaine solution. There was immediate relief from otalgia and vertex pain. On the next day Dr. Cone injected 1 cc. of SoluMedrol into the trigger area within the center of the upper edge of the trapezius muscle, without preparation with procaine. This was repeated after twenty-four hours.

She was symptom free when examined sixteen days later, on October 8, 1959.

There is no relationship between myofascial trigger areas within the postcervical area and pathologic changes in the mandibular joint which incite the mandibular joint syndrome. Such effects of impingement of skeletal upon sensory structures originate within the pathologic joint, resulting from malocclusion, jaw injury, mastoid infection, and other sources.

Procaine has been the most popular agent injected into painful myofacial areas, and it was shown here that the steroid substance is prompt and efficient without the use of procaine. Experimenting in clinic cases with mandibular joint neuralgias, we have found that injection of 1 cc. of serile water or saline solution into the joint space increased typical pain symptoms for two to four weeks, with increased trismus and at times glossodynia. Procaine or Xylocaine injection relieved pain in most cases for about one month, and these substances combined with hydrocortisone increased the period of relief from pain for periods up to one year.

Myofascitis (Albee), cervical somatic lesions (Kelly), the sternomastoid syndrome (Travell), the cervical syndrome (Jackson), the cervico-occipital syndrome (Missal) or any similar complexes by other names certainly deserve the interest of those who study headaches and sources of neuralgic pain.

OPPORTUNITIES FOR RESEARCH AND RESEARCH TRAINING IN THE FIELD OF HEARING

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THE establishment of the National Institute of Neurological Diseases and Blindness in 1950 was the direct result of recognition by the general public of the importance of the medical, social and economic problems caused by neurological and sensory disorders and signified determination to solve these problems. This recognition would certainly have been delayed many more years if it had not been for the efforts of a number of professional associations and voluntary organizations such as the American Academy of Ophthalmology and Otolaryngology.

I should like to review briefly the magnitude and nature of the public health problem leading to the establishment of the Institute, indicate the progress made in the last nine years, and point out opportunities for additional research and research training, particularly in the field of hearing.

As a distinct group of disorders, neurological and sensory diseases create an extremely serious problem, since they affect well over 20,000,000 people in the United States, of whom at least one half endure gravely disabling conditions. As a cause of death, neurological diseases rank third. However, it is as the primary cause of permanent impairment that neurological and sensory disorders are generally recognized. For example, cerebrovascular disease accounting for about 200,000 deaths annually also results in over 2,000,000 persons paralyzed in body or seriously limited in powers of movement, speech and vision. Other neurological and sensory disorders, similarly affecting the brain or spinal cord, are responsible for the disablement of an estimated additional 10,000,000 persons annually.

This crippling is usually so severe and extends over such long periods of time that it causes profound psychological, social and economic dislocation for the individual sufferer. Even today with increasingly large amounts of funds made available, no other group of disorders receives so little medical or research attention; yet no other group represents such a staggering economic loss to the Nation, to the patients, and to the families that must cope with them. This is the magnitude of the problem caused by such major disorders as cerebrovascular disease, epilepsy, cerebral palsy, hemiplegia, parkinsonism, aphasia, multiple sclerosis, muscular dystrophy, blindness, deafness and accidental injury to the nervous system.

In respect to the nature of the problem, research into neurological and sensory disorders has always been difficult. The nervous system is the most complex and widely dispersed system of the body. Until relatively recently it was the most inaccessible to study. In spite of its relative inaccessibility, however, it is particularly vulnerable to disease and injury. Moreover, central nervous system tissue, unlike other tissues, does not re-

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pair spontaneously when once damaged. Because of this characteristic, research must be aimed primarily at cause and prevention—an area where the signposts are relatively vague or nonexistent as compared to treatment, but, at least in some disorders, not quite so hopeless.

The magnitude of the problem of neurological and sensory disorders demands a concerted and continuous effort utilizing all available resources, both voluntary and governmental. The nature of the problem necessitates a broad attack, utilizing all medical disciplines and the various types of trained personnel. By joining forces with all interested agencies and organizations and by encouraging all possible approaches, considerable progress has been made in the last few years.

The first appropriation the National Congress made to the Institute for research in the field of neurological and sensory disease was for the fiscal year 1952 and amounted to $1,015,000. This sufficed to support 119 research projects, none of which were in the hearing and speech field. By 1955 the Congress had increased our budget for research grants to $4,000,000. In that year we supported 17 projects in the hearing and speech field in the amount of $175,000, which represented about 4.4 per cent of our budget.

These latter figures contrasted so sharply with the importance and magnitude of the problem of hearing and speech defects that a conference was called of the leading authorities in this field. At this meeting it became apparent that the few of the Nation's scientists competent to pursue research in this area were already doing about as much as possible and that any expanded effort depended on training additional investigators. In essence, for all practical purposes, this research field, personnel-wise, was almost bankrupt!

Subsequently, the Otolaryngology Postgraduate Training Committee was established to advise the Institute on the training of additional clinical and basic science research manpower. That this development, which will be described in detail later, has had some effect even in such a short time is evidenced by the fact that at present we support 74 projects in the hearing and speech field, involving a little over $1.3 million per annum and comprising 6.6 per cent of our budget.

Probably most noteworthy among these projects is such work as that being done at Johns Hopkins University on electrodermal responses associated with hearing under the direction of John Bordley; the work on conservation of hearing in children being carried out by Raymond E. Jordan and his colleagues at Pittsburgh under the guidance of the Subcommittee on Hearing in Children of this Academy; studies of the vestibular system by John R. Lindsay and Cesar Fernandez at the University of Chicago; work on laryngeal physiology by G. Paul Moore and Hans von Leden at the Gould Foundation; and Gordon Peterson's work on speech physiology at the University of Michigan, just to mention a few.

Certainly the research horizons in the hearing and speech field are broadening and financial backing is readily available to those who can present a proposal that seems to have potential for making a contribution. We are confident that the next few years will witness rapid development and increasing opportunity for those who are challenged.

The program of postdoctoral Training Grants and awards administered by the Institute of Neurological Diseases and Blindness was inaugurated in 1952. During the following twelve months, 16 grants were awarded for a total of $260,000. From this modest beginning

a little more than six years ago, this program for the advanced training of investigators in the broad field of neurological and sensory diseases has grown to its present proportions of 168 training program grants in the amount of $4,838,000 and involves the training of almost 900 individual trainees. In addition, a program of Special Traineeships has been developed which at present is supporting approximately 150 individuals for advanced training in various parts of the world at a cost of $1,250,000. Also, a program of Postdoctoral Fellowships is carried on at the $500,000 level. Thus, the Institute's total support for training at the present time is well over $6,500,000.

The first training grants to institutions were made in neurology and ophthalmology. The present program encompasses training in these two specialties as well as otolaryngology, neuropathology, pediatric neurology, neuroanatomy, neurochemistry, neuropharmacology, sensory physiology, and medical audiology. At the present time, therefore, the training grant program covers almost all neurological and sensory disease areas in which there is a recognized dearth of personnel trained for investigative work.

The purpose of all training grants awarded by the National Institute of Neurological Diseases and Blindness is to assist training institutions to increase the number of adequately trained personnel available for careers as investigators. Consequently, training grants are program grants made to help institutions establish, expand, and improve all aspects of their program of training in a particular field. Support is initiated on the premise that it will be continuous so long as the program is productive and a national need exists for the particular type of personnel being trained.

In the training of clinicians, funds are not awarded merely for the purpose of supporting the routine training of a house staff. Program Directors must show how financial support will be utilized so as to increase the potential for training clinical investigators. Translated into more practical terms, the Program Director is asked to outline how his proposed training program differs from that which would be adequate to train an individual for a private practice career. Only if the difference in approach seems to provide reasonable assurance of success is the grant made.

Training grants are awarded to supplement the training institution's own support of the program. Funds awarded may be used to employ additional staff, to provide additional trainee stipends, to purchase additional equipment and supplies needed for the training program, and to provide travel funds so that trainees may attend meetings and conferences. Since training grant funds are to be used supplementarily, the amount awarded to aid any program varies with the institution's need and the program's capacity to train.

Since the establishment of the Otolaryngology Postgraduate Training Committee, 23 programs have been inaugurated for the purpose of training clinical investigators. These 23 programs provide training for about 150 trainees. The Committee has also aided in the establishment of three programs in sensory physiology, primarily aimed at training basic science personnel, but also open to clinicians, and has pioneered in the establishment of two programs in medical audiology. These latter programs are for the purpose of providing medically oriented postdoctoral training to audiologists. It is believed that such training, carried out in a medical setting over a period of two or three years, will result in a product which, when teamed

with research-oriented clinicians, can maintain a balanced research effort in the hearing and speech field.

In terms of actual numbers of individuals concerned, present otolaryngology training grant funds aid in supporting 35 of the 85 professional staff engaged in training and 65 of the 150 residents involved in the 23 programs. As you can see from these figures, 60 per cent of the professional staff engaged in training duties and 60 per cent of the residents receiving training derive no financial aid from the funds awarded. On the other hand, all trainees benefit from the stronger program made possible by the award of Federal funds.

The support of house officers out of training grant funds is of sufficient importance to warrant detailed explanation of relevant training grant policy. It is the policy of the National Institute of Neurological Diseases and Blindness that the director of a training grant program should have freedom within the limits of his own institution's policies to choose the personnel he is to train and within a liberal stipulated maximum to set the stipend rate for those trainees to be paid in whole or in part from the training grant. These maximums, which are $4500, $5000, $5500, and $6000, plus $500 for each dependent for the first, second, third and fourth years, respectively, are frequently interpreted as mandatory. On the contrary, these rates are maximum levels which may not be exceeded without prior approval in each individual case. They were adopted to provide consistency with other types of awards made by the Public Health Service and other national granting agencies for the training of individuals at similar levels and with equal qualifications.

Since training grant funds are to be used supplementarily, stipends previously derived from institutional sources may not be transferred as an obligation to the training grant budget. Training grant funds budgeted for stipends may be used to increase institutionally established stipend rates, if such seems desirable and necessary. Such funds may also be used to increase the number of trainees by providing additional stipends. This point is stressed in all clinical training grants from the Institute because an increase in the number of trainees not only serves to increase the total research manpower, but also tends to lessen the purely service load on each trainee while in training. It is hoped that the time thus freed will be utilized for research training.

Since the objective of the grant is to train researchers, program directors are urged to support from grant funds only those trainees who are interested in or who seem to have a potential for investigative careers. The program director's choice of trainees is very important, because the only valid measure of the success of a training grant is the program's contribution from year to year in terms of individuals who choose investigative careers.

In contrast to training program grants made to institutions, wherein the institution chooses the personnel it is to train and sets the stipend to be paid, two other training programs provide direct Federal support to the trainee. The first of these—Postdoctoral Fellowships—are utilized by the Institute of Neurological Diseases and Blindness to provide support for the first three years of postdoctoral research training primarily in those disciplines or at those institutions not supported by training grants.

Postdoctoral Fellowship awards are made for training either in this country or abroad and are limited to United States citizens or to those who intend to become citizens. Stipends provided are $4500, $5000 and $5500, plus $500 for each dependent per annum for the first,

second and third years of training respectively, i.e., the same as the maximum stipend allowed under training grants.

Postdoctoral Fellowship awards to individuals are for the purpose of supporting training during the same period as Postdoctoral Training Grants to institutions but do not serve as a competing program. Postdoctoral Fellowships are usually awarded in those instances in which an individual wishes to train at an institution and in a discipline not covered by a program grant.

The Institute awards about 50 Postdoctoral Fellowships each year in fields ranging from biophysics to ophthalmic pathology. It is a necessary part of our total training program, since the establishment of programs of training supported by training grants in every conceivable subject and often for only one or, at the most, two trainees would not be justified.

The most advanced training awards made by the Institute are Special Traineeships. This program was established specifically to complement the Training Grant and Postdoctoral Fellowship programs. Special Traineeship awards are available to those who have completed training in a specialty or who have had not less than three postdoctoral years of training and experience pertinent to the additional training to be taken. Special Traineeship stipends may be awarded to the same individual for as long as five years and range from $6500 to $17,500 per annum. The specific amount is determined by the appropriate training grant committee on the basis of the applicant's qualifications and the cost of the training desired.

At the present time about 150 individuals are obtaining advanced training in this country or abroad under this program. The majority of these are neurologists, neuropathologists and basic scientists training in such fields as neurochemistry, neurophysiology, electron microscopy, and neuropathology. There are only a few, three I think, taking advanced training in the hearing and speech field. Hopefully, as more trainees receive research-oriented training at the residency level, and as the medical audiology programs start to produce, a larger number will be stimulated to continue until they can become competent investigators. It is obvious that thorough research training cannot be completed during the residency period.

In summary, this report has briefly touched upon the mission of the National Institute of Neurological Diseases and Blindness, how it attempts to carry out its function, and the opportunities for research and research training, particularly in the hearing and speech field. Suffice it to say that through a combination support derived from research grants, training grants, and individual awards, we can aid in the carrying out of research and in developing outstanding investigators in the field of sensory disease. As an additional aid we have just made a grant to the American Academy of Ophthalmology and Otolaryngology to finance an employment service for scientists in the sensory field.

It is generally accepted that a medical specialty or discipline cannot progress unless a considerable proportion of its representatives are competent investigators actively engaged in research. Although this circumstance does not prevail in either otology or audiology, the dearth of competent investigators is gradually becoming less acute. There is a long way yet to go. We hope that you will take full advantage of the opportunities provided so that future progress will be more commensurate with the importance and magnitude of the problem of hearing and speech defects.

DIFFERENTIAL DIAGNOSIS IN CHILDREN WITH COMMUNICATION DISORDERS: A FILM DEMONSTRATION

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THIS paper and the film which illustrates it are based on and inspired by an awareness of the ever-increasing number of children with central nervous system impairments who come to the attention of the otolaryngologist. These are the children that are being saved by modern medicine, those born prematurely or who suffered birth trauma, the anoxias, the infants with kernicterus and desperate cases salvaged with modern drugs, not to mention those whose troubles started in embryo in the early months of pregnancy when the mother developed some relatively mild virus infection or other disorder. In many of these children the first sign of difficulty is an impairment or lack of language development. Since they appear to be "deaf" and/or "mute," they are referred to speech and hearing clinics and to schools for the deaf. Because disorders of communication are usually the major problems under investigation in these children, the responsibility for assessment and differential diagnostic evaluation rests with a medical team, particularly the otologist and neurologist, and a paramedical team, particularly the speech pathologist and audiologist. Often the language problem is the only demonstrable manifestation of central nervous system impairment.

The assessment of children with communication disorders is based on the concept that language development is dependent on many general developmental factors, but especially the action and interaction of the peripheral sense organs and the central nervous system of the growing child.

The schema shown in figure 1 may be used as a frame of reference, indicating the major channels through which language flows.

In order to develop language normally, the child needs sufficiently intact peripheral sense organs of hearing as well as sight and touch to perceive stimuli (sensory data) from the outside world. He further needs sufficient intact central functions to interpret what he hears, sees and feels and thus to comprehend and use symbols. After symbols have been perceived and interpreted, perceived and apperceived, proper associations based on experience and learning have to be made in order to decide on an appropriate response within an appropriate time. To do this, the child must have a certain degree of intelligence. Moreover, since speech is a function of communication, he must also be sufficiently adjusted emotionally to want to communicate and he must live in a psychological climate conducive to communication.

From the Lester A. Hofheimer Speech and Hearing Clinic at the Columbia-Presbyterian Medical Center. Supported in part by the USPHS Grant OM 155 from the National Institute of Mental Health to the St. Joseph School for the Deaf.

Presented at the *Special Program* of the Otosclerosis Study Group and the Committee on Conservation of Hearing at the Sixty-Fourth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 11, 1959, Chicago.

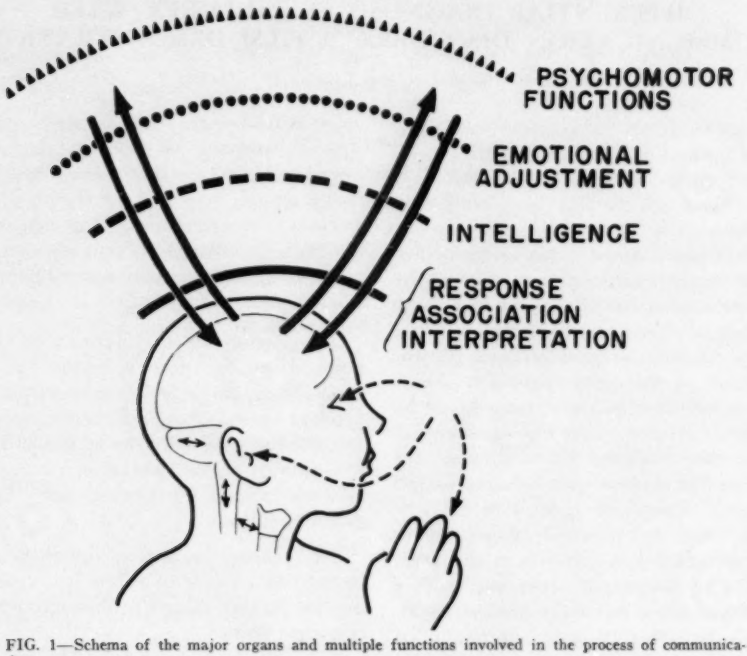

FIG. 1—Schema of the major organs and multiple functions involved in the process of communication.

In order to develop language, a child must develop psychomotor functions, which, on a cortical level, are responsible for his awareness of himself as an organism separate from the mother and from the world around him. He must have a concept of his own body and of spatial relationships, visual motor functions and the ability to focus on a stimulus presented. Only thus can he develop the ability to focus on an auditory stimulus, just as on any other specific stimulus, and perceive it as a whole against the background of other unstructured stimuli. In the words of Russell Brain:[2]

> The receptive function of the cerebral cortex is to provide us with a symbolic representation of the whole of the external world, not only distinguishing objects in their qualities but also conveying to us the spatial relationships which exist between them and at the same time giving us similar symbolical information about our own bodies and their relationship with the external world.

In evaluating disorders of communication in children, taking the peripheral, central and perceptual (psychomotor) functions into consideration, we try to determine at what point, or points, a breakdown or maldevelopment has occurred. This is done through observation and a battery of language and psychometric tests qualitatively used. After a considerable number of children had been thus evaluated, collation of the findings showed that many children with communication disorders who had been sent to us with a diagnosis of a peripheral hearing loss fell into different but none the less fairly specific pathologic profiles.[4] These profiles now serve as a basis for differential diagnosis.

The battery of tests and points of observation are now being used in a project designed to more clearly delineate children with communication disorders other than those caused by clear-cut peripheral hearing loss. The latter are being used as controls. The work is being carried out jointly by members of the staff of Columbia-Presbyterian Medical Center and Saint Joseph's School for the Deaf, both in New York. Several of these children have been selected for a cinematographic study to illustrate especially characteristic manifestations within the diagnostic categories. The aims in making the film were to demonstrate as clearly as possible the modes of behavior, the attitudes toward verbal communication, and the test responses characteristic of each pathologic profile, on the one hand, and to highlight the differences among the children in the entire group, on the other.

The purpose of the Saint Joseph's project is to test the reliability of our present diagnostic evaluation procedures and the value of our diagnostic terminology. We also expect to collect information which will serve to improve the training program for the children and offer a basis for better training of professional workers concerned with the welfare of these children.

Six representative children were selected for the film and this summary of it. They show different diagnostic entities found in children with communication disorders. Since behavior, changes in facial expressions and body movements cannot be transcribed from the film, the emphasis in the illustrations for this paper is on the reproductions of the Goodenough drawings of a man and the Bender Gestalt tests.[1] These also clearly indicate the organization in the normal child (figs. 2, 3 and 4) and the disorganization in the child whose central nervous system has been impaired.

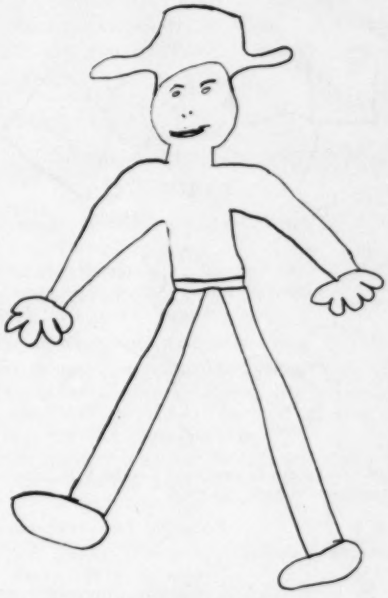

FIG. 2—Drawing of a man by R.B., a nonhandicapped 6:4-year-old child.

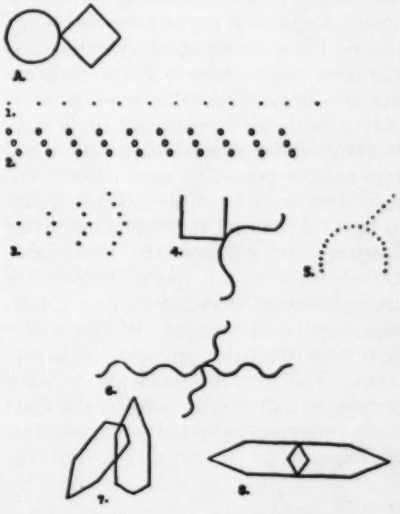

FIG. 3—Bender Gestalt Test Figures adapted from Werthheimer, which the children are asked to copy.

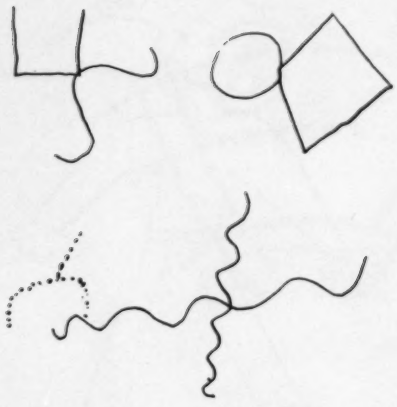

FIG. 4—Bender Gestalt Test done by R.B., a non-handicapped 6:4-year-old child.

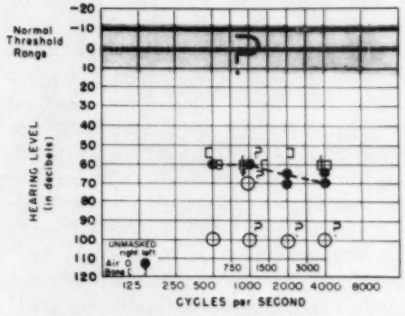

FIG. 5—Case 1, S.R. Best obtainable threshold responses for pure tones with head phones. The question mark reflects the audiometrist's opinion of the audiogram's validity. Downward pointing arrows on symbols indicate that there was no response at loudest intensity of BC available at that frequency.

Case 1

S. R. is a child initially diagnosed as having brain injury and central auditory imperception (dysacusis). Observing this boy, who is 6:5 years old, the impossibility of conditioning him to respond to pure tone audiometry is apparent. He is preoccupied with the material used (color cones). There is clowning and negativism. The impression of only a moderate impairment of hearing is obtained by clinical observation and free field responses to pure tones.* The responses with head phones are shown in figure 5. During the evaluation, using language and psychometric tests qualitatively instead of quantitatively, the same behavior dominates: i.e., clowning, negativism, teasing. In the course of testing, the basic problems come into focus. The deviant behavior is most prominent and overtly used by the child when responses to verbal communication are expected or requested. The boy cannot give proper responses, since he does not *comprehend* the spoken word he hears. The basic problem as well as the secondary defense mechanisms and compensatory patterns begin to crystallize. Thus, when responses which do not involve verbal speech are requested, the boy's behavior changes and cooperation is easily established. When the child places forms into their recesses (Seguin form board), jerky movements of the arms and hands can be observed, suggesting athetoid patterns. In drawing a man (Goodenough test) and copying

FIG. 6—Case 1, S.R. Drawing of a man. Note size of figure, although patient was as usual given a full-sized (8½ × 11 inches) sheet of paper on which to make his drawing.

*Sylvia Morgan, M.A., audiologist, tested the hearing in all cases reported. Her assistance is gratefully acknowledged.

designs (Bender Gestalt test), perceptual dysfunctions of body image and visual motor functions appear (figs. 6, 7).

FIG. 7—Case 1, S.R. Bender Gestalt Test.

In this patient, clowning, negativism, rejection of verbal speech or lip reading, lack of comprehension of spoken language, odd gestures, jargon, as well as perceptual dysfunctions and jerky body movements complete the clinical picture of a child with brain injury, consequent dysacusis, and possibly concomitant moderate hypoacusis (peripheral hearing loss).

The profiles for each of the major diagnostic categories in which this boy falls are given below.

Dysacusis (Central Auditory Imperception)

Profile 1

Symbol behavior: good for visual symbols (recognition)

Spatial orientation: attempted with aid of kinesthetic sense (feeling contours)

Visual motor functions: poor

Figure background perception: poor

Handedness: preference established

Communication: jargon; echolalia; perseveration; no lip reading; poor or no comprehension of spoken language

Poor auditory attention; better comprehension when attention paid; better comprehension for single words than sentences

Considered deaf by parents

Poor balance, poor coordination additional factors in some cases.

Hypoacusis and Brain Injury

Profile 2

Symbol behavior: poor

Spatial orientation: poor

Visual motor functions: poor

Figure background perception: poor

Handedness: no specific pattern

Communication: pantomime; little attempt at lip reading

Voice: no specific pattern

Short attention span

Hyperactivity; motor disinhibition

Temper tantrums; bed rocking

Case 2

The second child, T. C., a girl of 5:4 years, with the initial diagnosis of post-encephalitic expressive aphasia and perceptual dysfunctions presents another picture. Conditioned responses to pure tones are easily established and a reliable audiogram can be obtained (fig. 8). The contrast with the first child in behavior during the language and speech evaluation is equally apparent: here is a girl who shows, in facial expression and responses, her concentration on the task, her desire to cooperate and her use of gesture, pantomime and facial expression to replace her basic dysfunction, inability to use words. This child clearly shows impaired perceptual functions, as can be seen in figs. 9 and 10.

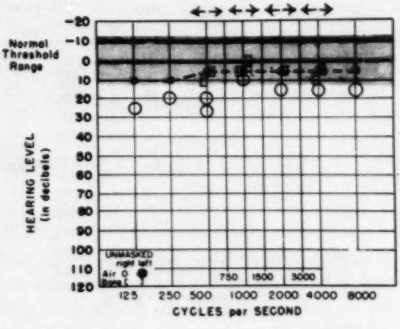

FIG. 8—Case 2, T.C. Pure Tone Responses, Weber, not lateralized at any frequency (indicated by arrows).

APHASIA, Acquired, Expressive

Profile 3

Loss of speech after at least some normal development reported
Symbol behavior: poor (anomia)
Spatial orientation: poor
Visual motor functions: poor
Figure background perception: poor
Handedness: use of better hand
Communication: jargon, gestures, some perseveration
Voice: good
Poor imitative behavior, including speech
Restless, hyperactive.

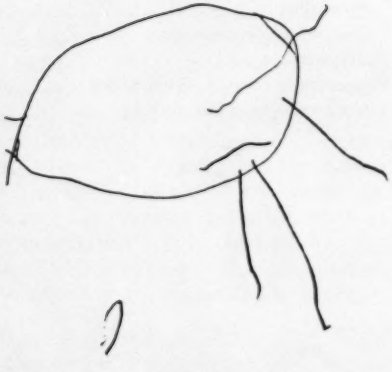

FIG. 9—Case 2, T.C. Drawing of a man.

FIG. 10—Case 2, T.C. Bender Gestalt Test.

Case 3

With D. P., a girl aged 6:10 years, cooperation for an audiogram could not be obtained. Her behavior shows clearly rejection of contact with children or adults, hostility and aggression, and a self-imposed withdrawal from the group. Her facial expressions reflect loneliness and unhappiness and the manifestations listed in Profile 4. This girl was eventually diagnosed as having emotional disturbance as the major cause of her lack of language development. The major problem with children in this group is to ascertain whether the emotional problem is primary or secondary. Stormy emotional overlays are the rule in language-impaired children and in their parents, whose marriages often break down.

MENTAL ILLNESS

Profile 4

Rejection of test performance
or
Symbol behavior: good
Spatial orientation: good
Visual motor functions: poor
Figure background perception: rarely elicited
Handedness: ambilaterality or use of both hands at once.
Communication: no response to spoken language; no lip reading; no attempt at communication; may take hand of parent or adult to point or take food and objects.
Voice: shreiks
Odd postures of hands: preoccupation with own hands
Lacking contact with people or environment
Good motor development and functions (climbing, jumping)
Reported love for music: singing, often with words in the absence of speech
"Enthralled by music"; "Never wants anything"; "Always been a quiet child"; "Always on the fringe"; "No contact with children"; "Ignores people"; "Lives in a world of his own"; "Lives with his hands"; "Shows no sign of pain".
Mechanically inclined: compulsive interest in wires, locks, etc.
Parents know the child can hear.

Case 4

G. E., a girl, 6:5 years old, obtained initial diagnosis of dysacusis and organicity with emotional overlay. This girl now shows excellent cooperation: she can easily be conditioned for pure tone audiometry (fig. 11). During the language evaluation, the discrepancy between her ability to respond to visual as compared to auditory symbols is striking, showing her lack of comprehension of spoken words, which she repeats but uses in response without understanding what she is saying (echolalia). Considerable emotional involvement manifested by masklike staring, talking and grimacing when spoken to, peculiar hand movements and body postures as well as alternate hypermotility and rigid withdrawal was present initially, but reduced markedly as soon as training started. Her perceptual functions are abnormal, as the drawing of a man and the copy of the Bender Gestalten show (figs. 12 and 13). Her diagnostic profile is Profile 2.

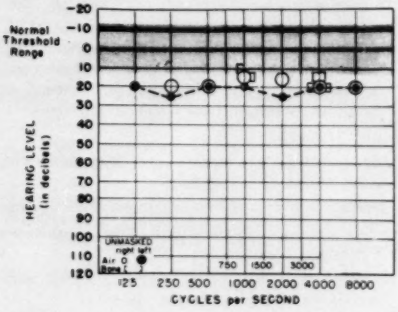

FIG. 11—Case 4, G. E. Audiogram.

HYPOACUSIS AND BRAIN INJURY

Profile 2

(See p. 533.)

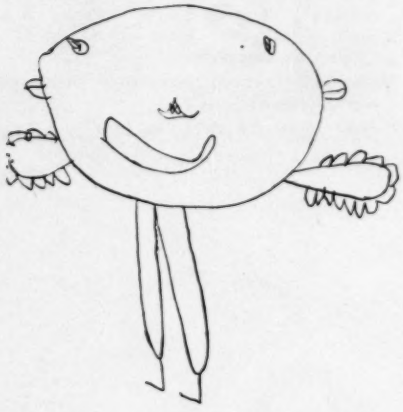

FIG. 12—Case 4, G.E. Drawing of a man. Very primitive for a child of this age.

FIG. 13—Case 4, G.E. Bender Gestalt Test.

Case 5

G. P., age 2:10, is the younger brother of this last child. Not only is he the second sibling in a family of three children to have impairment of communication, but his behavior reflects, to a greater extent than his sister, a personality disorder as well as a language disorder. Observing this handsome, blond little boy one can readily see his lack of response to any stimulus, auditory, visual, or tactile. He too cannot be conditioned to respond to pure tone stimulation and the only indication of his hearing level is obtained free field and through observation when he occasionally responds to loud sounds and to his name called in a loud voice (fig. 14). His only display of interest is for mechanical objects and movement. He will reach for the microphone and finger it; he places the Binet forms quickly and correctly, but ignores pictures and shows little interest or use of objects such as a toy shoe, a cup, spoon or toothbrush. He handles the color cone well and shows good manipulating ability. However, as soon as the cone is turned or he turns it, both his hands go up to the sides of his ears in a fluttering movement, which is maintained while the toy is spinning. It is interesting to observe that during the entire interview this young child does not once look at, or touch, his mother who sits beside him. Nor does he turn toward her when under stress—he turns away from her, during the fleeting moments when he shows affect (distress). Thus, he shows many features found in both Profile 4 and Profile 5.

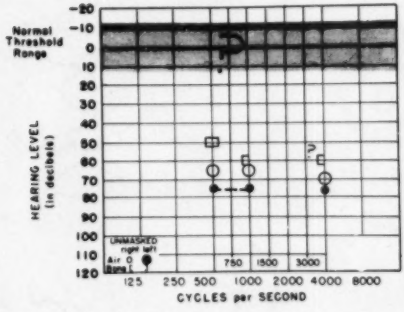

FIG. 14—Case 5, G.P. Best obtainable pure tone responses but question mark indicates that the levels of response of each frequency are probably not at threshold.

MENTAL ILLNESS

Profile 4

(See p. 535.)

HYPOACUSIS AND EMOTIONAL DISTURBANCE

Profile 5

Symbol behavior: good

Spatial orientation: good

Visual motor functions: good

Figure background perception: fair

Handedness: no specific pattern

Communication: verbal, defective articulation; little attempt at lip reading

Voice: whisper

Child appears withdrawn but can be reached; difficulty with peers reported

Case 6

Contrary to the other children, B.J.A., age 5:8 years, was diagnosed as having simple anacusis (deafness greater than 82 db level for the speech frequencies) (fig. 15). Her expression, her responses and functions clearly indicate the "normalcy" of her reactions and show a child who is anxious to communicate, knows exactly what she wants to communicate, and uses lip reading and all possible means to comprehend and to express herself, despite the severe hearing impairment. Her body image and visual motor functions, however, are not up to par, yet are qualitatively different from the organic, disorganized child (figs. 16, 17); a frequent finding in anacusic children as shown in Profile 6. Compare the drawings of a nonhandicapped child (figs. 3 and 4).

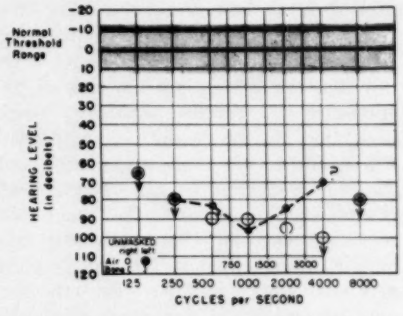

FIG. 15—Case 6, B. J. A. Audiogram. Downward pointing arrows indicate frequency was not heard at loudest AC intensity available on the audiometer used.

FIG. 16—Case 6, B. J. A. Drawing of a man.

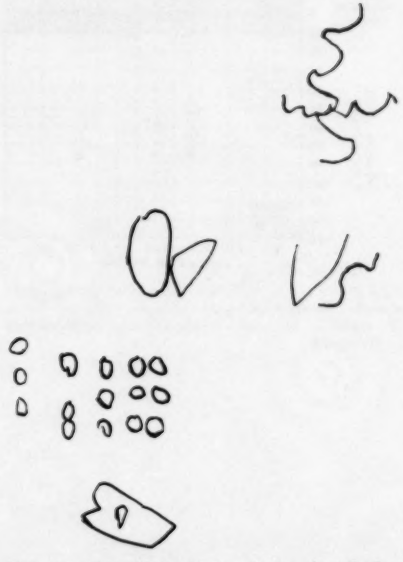

FIG. 17—Case 6, B. J. A. Bender Gestalt Test.

DEAFNESS (ANACUSIS)

Profile 6

Symbol behavior: good (visual and tactile)
Spatial orientation: good
Visual motor functions: fair
Figure background perception: fair
Handedness: preference established
Communication: pantomime; lip reading (spontaneously)
Voice: flat, "deaf"
Poor responses even to strong auditory stimuli
Parent able to state when hearing impairment was first apparent; child could detect airplanes and slamming doors (probably by vibration).

The study of this and other groups of children with communication disorders has pointed to the need for thorough differential diagnostic evaluation of children who seem to have a hearing impairment but do not respond appropriately to the routine testing or training for a child with anacusis. These are the children who only *seem* to be "deaf" and/or "mute." The sooner comprehensive diagnostic evaluation is instigated in the life of the language impaired child and the earlier his training is started, the clearer the pathological profile, and the more successful the rehabilitative program (figs. 3 and 5).

REFERENCES

1. Bender, Lauretta: A Visual Motor Gestalt Test and Its Clinical Use, The American Orthopsychiatric Association, New York, L. G. Lowrey, 1938, p. 4.
2. Brain, Walter Russell: The Nature of Experience, London, Oxford University Press, 1959.
3. Hardy, William G.; Pauls, Miriam D., and Haskins, Harriet L.: An analysis of language development in children with impaired hearing, Acta oto-laryng., Suppl., 141:1-51, 1958-59.
4. Kastein, Shulamith, and Fowler, Edmund P., Jr.: Differential diagnosis of communication disorders in children referred for hearing tests, A.M.A. Arch. Otolaryng., 60:468-477 (Oct.) 1954.
5. ———: Language development among survivors of premature birth, A.M.A. Arch. Otolaryng., 69:131-135 (Feb). 1959.

I.

THE HISTOPATHOLOGY OBSERVED IN FIFTY BIOPSIED STAPEDES

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THE stapes, the smallest and most fragile of the 201 bones of the body, has commanded the attention of investigators for three and a half centuries. This stirrup-shaped bone has a footplate which is 1 x 2 mm., and the distance from the footplate to the top of the head is a mere 3 mm. According to Anson and Bast, the specific gravity of this ossicle is so slight that it displaces only 0.2 cc., or two or three drops, of water.

Formed in the fifth week of embryonic life as a cartilaginous ring around the obturating second aortic arch or stapedial artery, this structure is gradually developed into the two curving bony crura which rise from the bean-shaped footplate and culminate in a neck and head of variable size and density. The mature size is attained in the eighth fetal month.

The stilted arch thus created is surmounted by the neck and head of the stapes, which form the crown of the arch. In the fifth fetal month the hematopoietic marrow, hitherto contained within the crura and above the intrados or soffit of the arch, is resorbed. This leaves a curving groove which is bound by a shell-like layer of lamellar bone of variable thickness. The bone is covered by intimately applied mucoperiosteum, which in its normal state can scarcely be discerned under the microscope, except where capillaries traverse it.

Normally no osteons or haversian systems are present in the lamellar bone of the crura. In horizontal sections of the temporal bone, cross sections of minute arterioles can be seen at the base of each crus. These divide into from two to five branches, one or more of which pass through the mucosa up the depths of the hollow groove of the crus on each side, meeting in a plexus beneath the intrados of the arch. Nager and Nager[13] found that the blood suppy for the stapes is derived from three sources: (1) the superficial petrosal artery (a branch of the middle meningeal) which travels in the facial canal and emerges to supply the posterior part of the stapes; (2) the superior tympanic artery, which supplies the anterior crus; (3) the inferior tympanic artery, which approaches the bone anteriorly, to supply the base.

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Great variability in the structure of the head and neck of the stapes as well as in that of its crura has been described by Bast and Anson, Farrior, Rosen, Hough, and others. Not only the solidity and shape of the head and neck may vary but also the degree of curvature of the crura. Usually the posterior crus is more curved, but instances in which the anterior crus was more bowed have been observed by us.

The present study deals with the pathology of this minute bone. It is based on microscopic sections from biopsied specimens collected from patients upon whom a mobilization had been performed for otosclerosis. The survey includes fifty stapedes collected from the year 1954 to July 1959. The ages of the patients ranged from 10 years to 66. Six patients were in the second decade of life; fifteen, in the third; thirteen, in the fourth; thirteen in the fifth; and two in the sixth.

The incidence of the sexes in the different decades may be worth noting. The 10-year-old child was male. In the second decade, four were female and two were male; in the third, eight were female, five were male; in the fourth, nine were female, four were male; in the fifth, nine were female, four were male; in the sixth decade one was female, one was male.

All ossicles studied showed evidence of pathologic change in the bone. Figure 1 illustrates the cross section of a crus from a male patient, aged 49 years, with a clinical history of "deafness"* for thirty-four years. In spite of this long history of the disease, the ossicle is still viable. Some lacunae are empty but others contain large osteocytes. The bone shows marked metachromatic staining. The mucoperiosteum lining the groove has become thickened and hypervascular. In fact, a sizable and thick-walled arteriole travels toward the head, in the depths of the groove. It should be noted here that marked congestion and stasis were present in the other two ossicles, which were also brought to the laboratory.

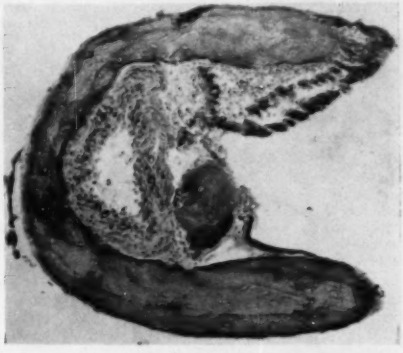

FIG. 1—Male, 49 years old. Cross section of crus from an otosclerotic stapes. Note uneven staining of the bone, the empty lacunae and the irregular contour of the inner wall of the groove. Within the groove observe the enclosed arteriole covered by mucoperiosteum. The nodule of bone is an artefact, having fallen there at the time of removal or during processing.

A series of three stapedes were cut longitudinally, so that the hollow groove of the crus was clearly seen in each (fig. 2). The first is the crus opposite the one from which the specimen shown in figure 1 was taken. Here again we see metachromatic staining. The center of the groove is filled with red blood cells and serum. The second specimen shows the hollow beam structure in a more atrophic bone, although it comes from a younger person (33 years old) and one who has not been deaf so long (eleven years). The third specimen presents the hollowed groove of a crus in an even more necrotic and atrophic bone. It was taken from a man 50 years old, who had complained of deafness for only one year.

*The words *deafness* and *deaf* as used in this paper are the descriptive words given by the patients at the time the case history was taken by the examining physician.

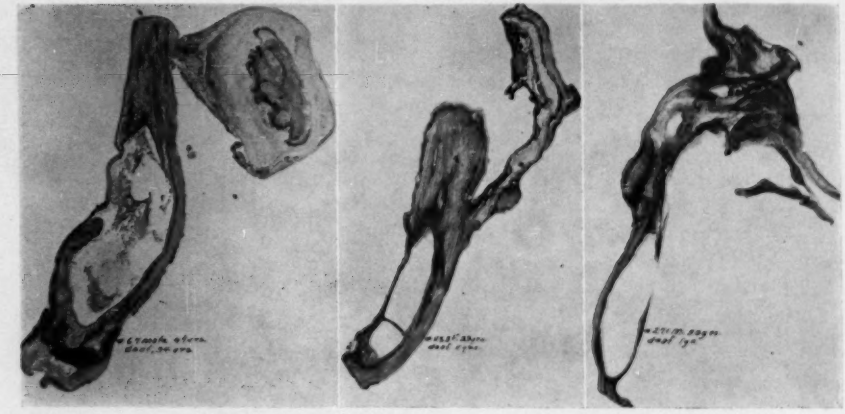

FIG. 2—Three specimens, each illustrating a longitudinal cut of a crus, showing the hollow groove in each. Mucoperiosteum lining the groove is intimately applied to the bone. Head of stapes has been dislocated in each of the first two specimens. In the third it is *in situ* and the lenticular process is also *in situ*. Thus an atrophic joint with fibrosis and early ankylosis with the incus is revealed. This patient has complained of "deafness" for only one year. At operation, the stapes was found fixed to the promontory. The stapes is osteoporotic and extremely fragile throughout. It is probable that sound entered the inner ear until fixation to promontory occurred.

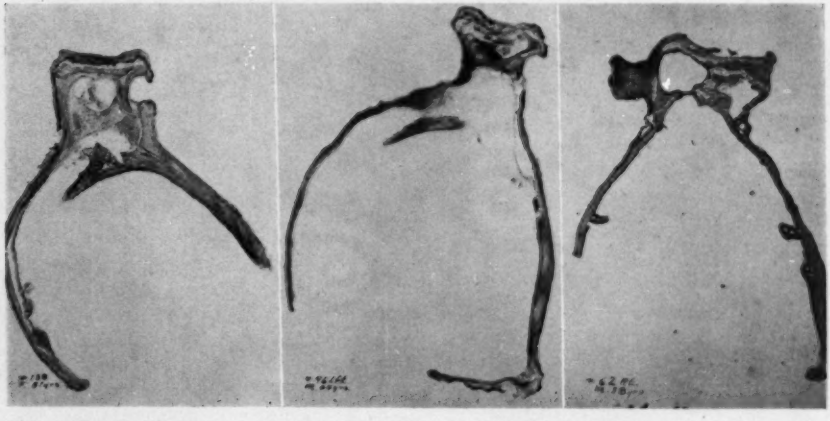

FIG. 3—The fragility of the stapes in some cases of otosclerosis is clearly depicted and exemplified in these three specimens, two from males. The first two are from left ears, the third from a right ear. In the first case the patient is 51 years old; the second, 44 years; the third, 38 years.

The most common type of pathologic change observed was extreme fragility of the stapes (fig. 3). This condition must not be casually judged from a single section, however. The interpretation of fragility, based on microscopic sections, must be made from a study of serial sections, because of the hollow-beam structure of the crura, described above. Therefore, each stapes was cut serially

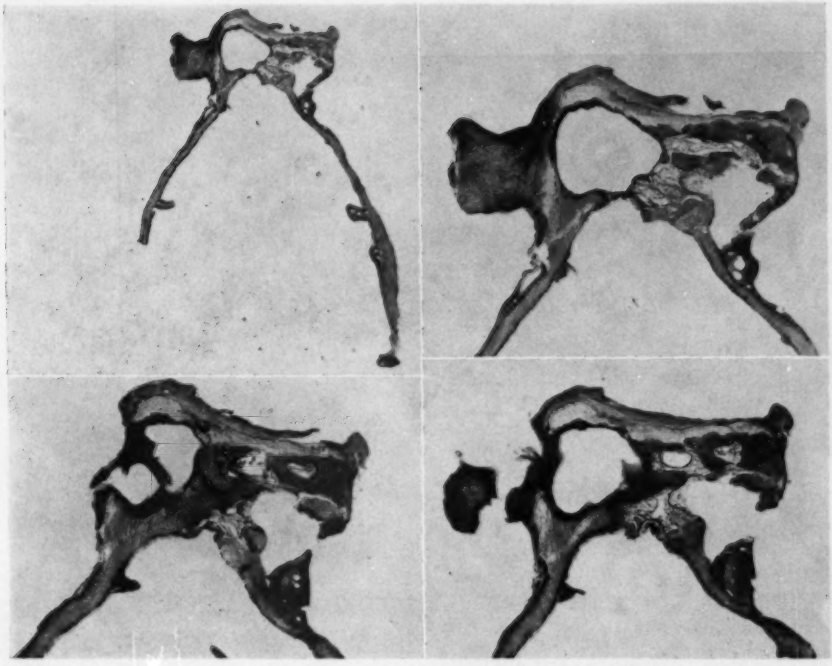

FIG. 4—Male, 38 years, "deaf" for 5 years. At operation, advanced otosclerosis at anterior end of footplate. Serial sections reveal a necrotic cystic type of degeneration throughout the head and neck.

and stained. With the celloidin technique it is not always possible for the technician to mount serially, but perfect sequential order may readily be ascertained from photographs. Furthermore, fragility may be manifest in a number of ways other than an extremely slender pair of crura.

Fragile stapes usually results from a loss of blood supply. The middle stapes in figure 3 is particularly interesting because we see that the footplate was also fragile. In the stained sections, this footplate exhibited three minute circular bodies on the inner surface which stained pink in hematoxylin and eosin. They are minute capillaries filled with necrotic red cells which have undergone amyloid degeneration. Normally there are no blood vessels on the vestibular surface of the stapes. This patient is 44 years old and had been "deaf" fourteen years prior to the operation.

Serial sections through the head of the third fragile specimen illustrated in figure 3 are shown in figure 4. They come from a 38-year-old male who had been "deaf" five years. Note that the bone in the head and neck has been eroded up to the cartilaginous articular surface. The connective tissue, where present, is necrotic and contains widened necrotic vascular channels. These are for the most part devoid of content, as are, likewise, the lacunae. Observe that the anterior crus (identified readily by the presence of the tendon attached to the poste-

FIG. 5—Male, 58 years old, "deafness" for 46 years. Left ear. At operation the footplate was found entirely replaced by otosclerosis. Microscopic sections show fragile neck and head. Fibrotic replacement of bone extended through to the articular surface. Note canalization of remaining bone and empty lacunae.

rior aspect of the neck) is fractured just below the neck.

The process of forming a fragile head and neck is well exemplified in a specimen from a 58-year-old man who had been "deaf" forty-six years, the longest history of "deafness" in the group (fig. 5). The only solid bone remaining is canalized with numerous vascular channels, now devoid of content. Note also that connective tissue replacement of bone has extended to the articular surface. Within the hollow neck of the stapes lie masses of necrotic red blood cells. Evidence of stasis in the mucoperiosteum, as well as petechial hemorrhages therein, may be seen in the lower left field. It seems apparent that the vascular plexus in the mucoperiosteum at the top of the arch of the crura may become a stagnant pool of blood, fed from the vessels coming to it from the groove of the crura, but ineffectively drained therefrom. While architecturally the bone structure is recognized as the strongest possible type of arch, its durability is vulnerable to degradative chemical substances locally-formed, apparently as a result of stasis. Perhaps the stagnant blood becomes acid and causes the calcium phosphate crystals to slip into solution. As this occurs, the freed osteocytes revert to fibroblasts which build up fibrous tissue to replace the bone. This process does not halt resorption.

Figure 6 illustrates vividly four different types of fragility in the head and neck of the stapes. The first specimen is from a female patient, aged 53 years, who has been "deaf" ten years. The bone in the head and neck is eroded to the articular cartilage. The borders of the gouged-out remnants of bone stain more intensely than normal with hematoxylin

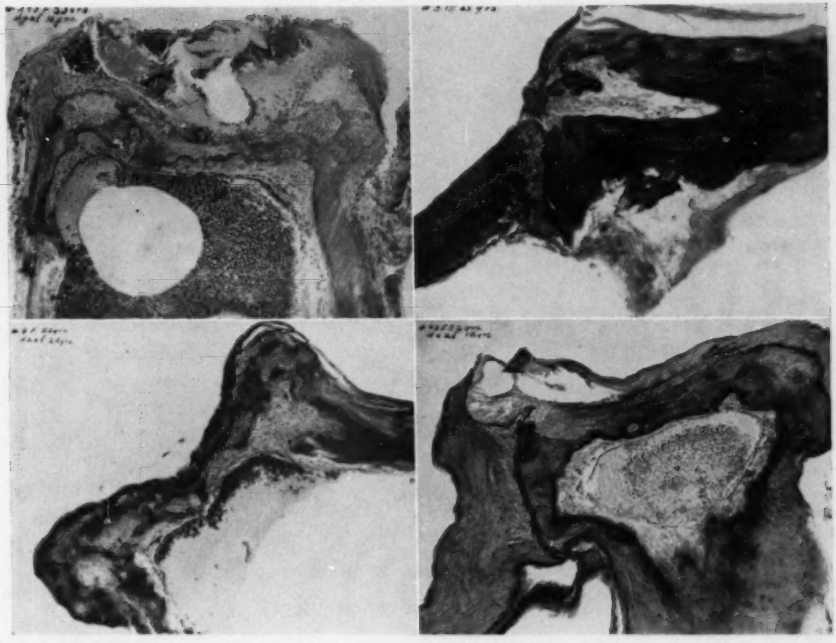

FIG. 6—Pathologic changes in the heads of four stapedes. The gouged out areas on the under surface of the head present the process of decalcification and fibrotic replacement of bone. Staining reactions around these areas are often intense, as in blue mantling. This is evidently a "chemical indicator" of a change in the composition or chemistry of the bone. On the right we see a normal tendon attachment above and cystic degeneration with liquefaction, below.

dye. This great affinity for the dye reminds us of the differential metachromatic staining of bone in the experimental cats upon application of an electric cautery needle. In this instance, however, we do not find blood vessels in the bone around which the decalcifying process is occurring. It is, rather, occurring around individual osteocytes, hence the gouged contours. We also observe that the articular cartilage in this specimen is undergoing liquefaction, with evidence of the nuclei streaming as in a liquid medium.

Below this photo we have a similar but more intensely stained specimen. Again the bone is gouged out or "moth-eaten" in appearance, sometimes leaving islands of calcium in the connective tissue. The freed osteocytes are conspicuous in this case, and the reaction of the bone to the dye certainly suggests a "chemical indicator" of the degree of dissolution. A calcium plaque has been deposited on the articular surface.

Above and to the right we see a more solid stapedial head with extensive fibrotic replacement near the attached tendon. Below is a view of another case showing cystic degeneration at the point of attachment of the tendon. This patient has been "deaf" eighteen years. Of course we do not see the microscopic picture of the stapedial muscle. Very

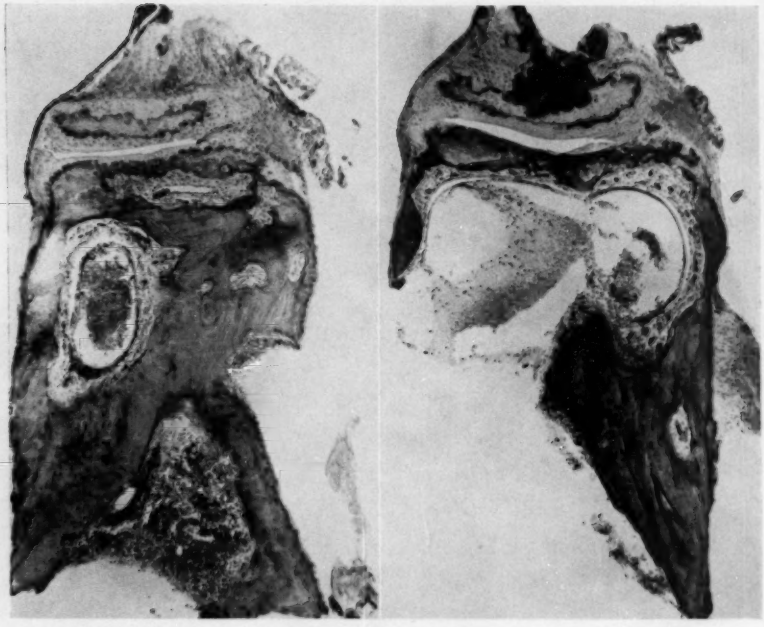

FIG. 7—Sections from two levels of a stapes from a man 23 years old, "deaf" for three years. Early degenerative changes of the incudostapedial joint with fibrosis and deposition of flecks of calcium are seen. Note the descending "chemical flow" of calcium from the bone of the incus into its articular cartilage. Hypervascularity, congestion and stasis are present in the mucoperiosteum.

probably it had become atrophic and this degeneration is reflected in the attachment of the tendon, a part of atrophy of disuse.

Occasionally the descending process of the incus is so fragile that this bone fractures, leaving the joint capsule intact, thus giving us a chance to study the degenerative changes in the joint. A relatively early stage of degeneration in this area is demonstrated in figure 7.

This specimen was taken from a 23-year-old male who had been "deaf" three years. The posterior portion of the joint capsule shows fibrosis and early deposition of flecks of calcium. The vessels in the mucoperiosteum of the head and neck exhibit marked congestion and stasis. The calcium in the descending process of the incus gives a picture suggestive of a liquid state and "chemical flow" which is usurping the area of the articular cartilage. These conditions would eventually lead to ankylosis.

Marked contrast in joint disease is seen in figure 8. The first specimen shows atrophy of the head of the stapes with erosion of the articular cartilage, one area being completely denuded. This is in a 33-year-old woman who had been "deaf" eleven years. Comment will be made later in regard to the fracture in the anterior crus. Another example of atrophic joint was shown in the third specimen of figure 2. There ankylosis, both fibrotic and bony, had occurred. This would give the ossicles a columella-like structure and may possibly explain

why this man of 50 years had a hearing difficulty of only one year (the shortest period of "deafness"). The ankylosed incudostapedial joint may have been more effective in the presence of a diseased footplate than a normal incudostapedial joint would have been.

The second photograph in figure 8 shows a large head extensively replaced by fibrosis and the articular cartilage marred by cystic degeneration. Within each cyst is granular particulate matter. Liquefaction of the cartilage is present. We know that hyaluronic acid is a normal component of joint fluid. Obviously the normal fluid has undergone some chemical change. The specimen comes from a woman 40 years old who had been "deaf" twenty-three years. In surgery the bone appeared pale and white. This seems to be the gross aspect when the microscopic sections show fibrotic replacement of bone.

We have mentioned fracture in the microscopic sections. Figure 9 is a surface view of the outer wall of the neck of a stapes from a 51-year-old woman. She stated that she had been " 'deaf' several years." A fracture line extends across this bone and calcium has been deposited in the crevice. Successive serial sections beyond this level revealed that the calcium deposition extended as a thin plate of bone for some distance into the connective tissue within the hollow neck of the stapes, appearing in successive sections as a thin bar of calcium. Nevertheless, a true callus cannot be said to have formed. We have no way of knowing how recent this fracture was, nor whether, with time, Nature would have succeeded in building a true callus. In figure 8, photograph 1, no healing has occurred in a fractured crus in a highly atrophic and necrotic bone, although the dark stain on the lower segment indicates some reaction to the injury. The

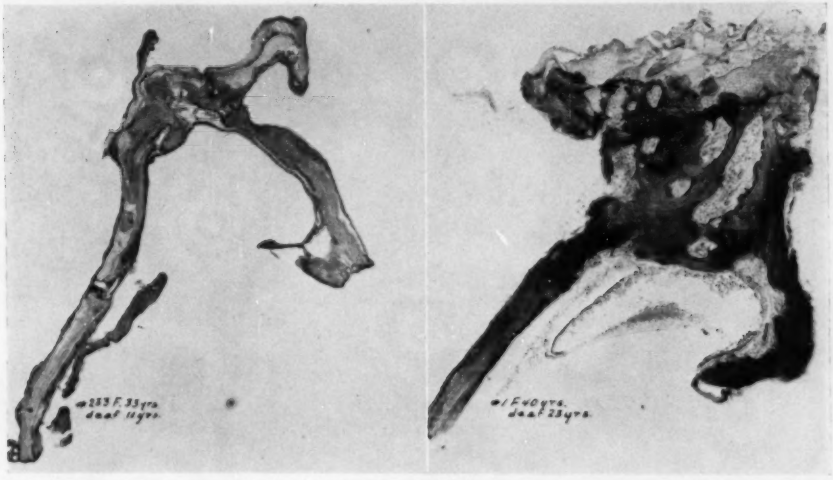

FIG. 8—Marked contrast may be seen in the pathologic changes in these two stapedes. On the left a fracture, unhealed, is present in the crus. The head is necrotic and fragile. The history stated that the stapes had refixed after mobilization and a polyethylene tube was inserted. On the right the specimen shows a relatively large head with extensive fibrotic replacement of bone. The joint capsule exhibits cysts containing granular particulate matter. At operation the footplate appeared white and was entirely replaced by otosclerosis.

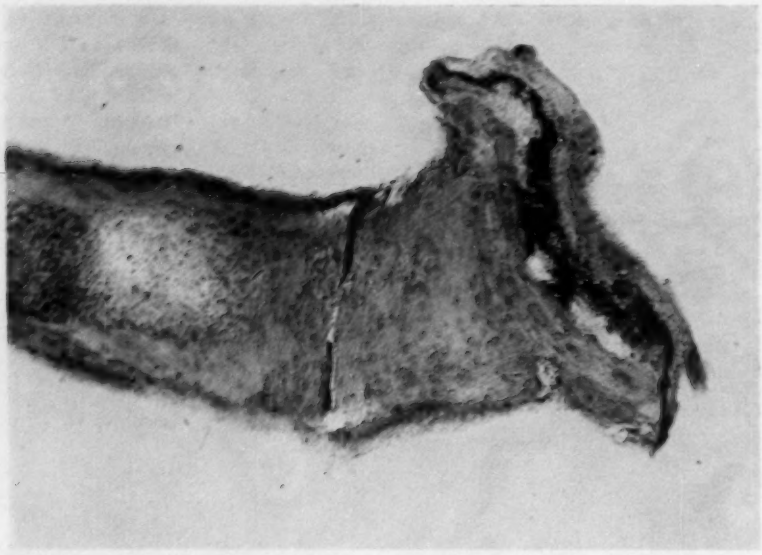

FIG. 9—Left ear of a woman, 51 years old with "deafness for several years." Although the neck appears to be quite solid at this level, note a fracture line with calcium deposited in the crevice. Deeper sections revealed a hollow, fragile neck and head.

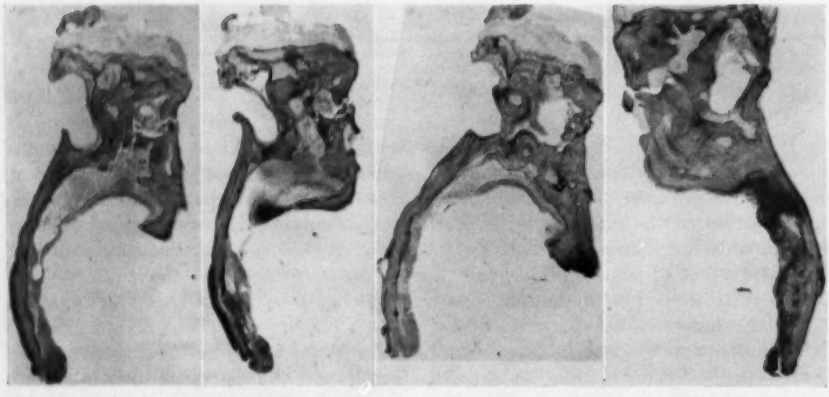

FIG. 10—Male, 49 years old, "deaf" for nineteen years. At operation, oval window was found to be reduced in size and the footplate fixed and replaced by otosclerotic bone. Microscopic sections reveal liquefaction of articular cartilage in incudostapedial joint, marked erosion of bone in head and neck, trapped serum and red blood cells beneath neck at point of beginning erosion. Both crura are involved in the formation of pathologic bone.

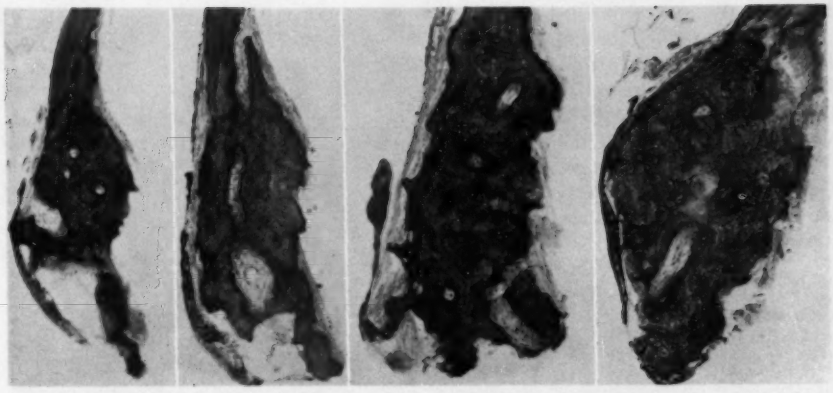

FIG. 11—Male, 35 years. One otosclerotic crus, from different levels. Note hypervascularity with some perivascular fibrosis, large lacunae with viable osteocytes and markedly irregular contours of the bone. No normal alignment of osteoblasts occurs. In the lower part of photo 3, enlarged fibroblasts are becoming encased in calcium. Calcium is being deposited on the outer periphery of the crus by accretion, an entirely different process.

FIG. 12—Massive overgrowth of anterior crus with ankylosis to vestibular wall and involvement of footplate. From a man of 50 years who had been "deaf" twenty years. Many viable osteocytes are still present in this bone.

specimen is from a 33-year-old woman who had been "deaf" eleven years.

A much more conspicuous type of pathologic change is the overgrowth of bone frequently seen in these cases of otosclerosis. In a series of sections from the stapes of a 49-year-old male who had been "deaf" nineteen years (fig. 10), the massive head and neck as well as the crura are in great contrast to some specimens already presented. Calcium has been deposited by accretion along both surfaces of one crus. The other crus is massively overgrown with pathologic bone deposited around two blood vessels, as if in an attempt to form two haversian systems or osteons. We must think of this type of pathologic change as distorted chemical crystallization and not as bone growth, in the sense of growth to maturity.

Four sections from one crus of a 35-year-old male exhibit hypervascularity, fibrotic replacement of bone and viable bone cells within enlarged lacunae (fig. 11). This pathologic process is in an active state. There is, however, no alignment of osteoblasts. In the third illustration of the series, it appears that individual fibroblasts have become enlarged

and have gradually deposited the calcium phosphate around them. Thus each cell becomes imprisoned within a lacuna of its own construction. At the same time calcium is being deposited by accretion on the opposite side of this same crus. This seems to be a somewhat different type of chemical "salting out," no cell actually taking part in the deposition as an individual unit.

A third example of enlarged bone is seen in the stapes of a man 50 years old who had been "deaf" twenty years (fig. 12). Here again we see hypervascularity of the bone in the anterior crus with extensive overgrowth of an amorphous mass and evidence of ankylosis of the thickened footplate to the vestibular wall, a fragment of which appears in the section.

A fourth example of massive enlargement of bone was found in the stapes of a 28-year-old woman who had been "deaf" eighteen years (fig. 13). An operation disclosed a vascular type of lesion. Some of the sections showed congestion

FIG. 13—Female, 28 years old, "deaf" for 18 years. At operation a vascular lesion was found replacing footplate and crura. One pathological vessel ascends the normal bone of the crus. Lacunae have no cells.

FIG. 14—The greatly thickened and hypervascular footplate from a 53-year-old man. Metachromatic staining is present.

and stasis, but here (fig. 13) the vessels are atrophic and empty. This would lead to an osteoporotic type of bone. Probably an embolus formed to cut off the blood supply to this area after the hypervascularity had developed. It is apparent that the lesion is extending into the normal bone along a blood vessel.

be greatly reduced in size, so that the niche and footplate were entirely replaced by otosclerotic bone. This appeared to be excessively vascular, and during the operation it bled profusely. Microscopically the tissue shows evidence of the hypervascularity, with stasis and congestion. Metachromasia is

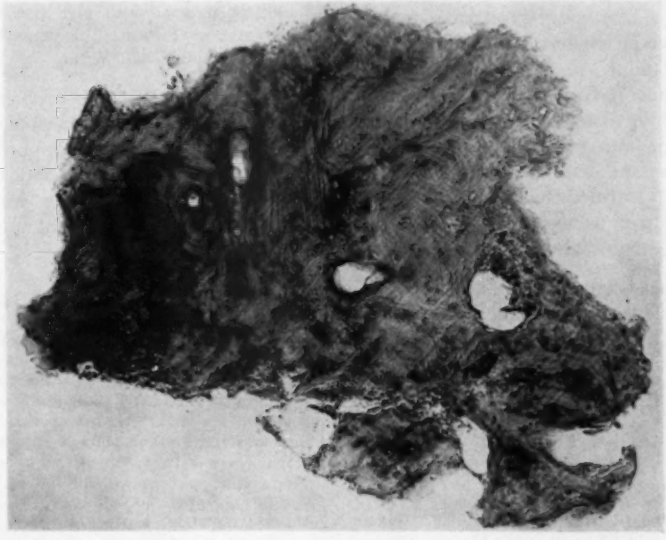

FIG. 15—A typical mass of otosclerotic bone removed along with five other similar fragments from the walls of an oval window, much reduced in size from the disease. Age of patient, 46 years. "Deafness," 20 years. Female, left ear.

A fifth example of enlargement of bone is seen in the footplate from a 53-year-old man (fig. 14). Note the hypervascularity with a tendency to form perivascular connective tissue. There are abortive attempts to form haversian systems and there is evidence of metachromatic staining.

The final illustration is a mass of otosclerotic bone removed with five other fragments from a 46-year-old woman who had been "deaf" twenty years. At operation her oval window was found to

conspicuous. Some of the other fragments showed necrotic mature bone; others showed viable active otosclerosis like this specimen.

In general the types of pathologic change seen in this material follow closely the types observed in postmortem studies of otosclerosis. They also correspond to that found in biopsied malleus and incus. Microscopic findings correlated well with observations at surgery. When the surgeon reported seeing a white bone, the sections usually showed

fibrotic replacement of bone. When it appeared red and injected, the tissue showed hypervascularity. Atrophic states could not be correlated with the age of the patient nor with the duration of the disease. We never found osteoblasts or osteoclasts.

Even in a bone as tiny as the stapes this lesion may be highly localized. It seems possible that the decalcifying agent may be some degradative enzyme, locally produced. Biochemists have identified alkaline phosphatase in tissue where bone is forming. It is said to be produced by osteoblasts. "Showers" of calcium or bone salt have been described as the precipitate. An acid phosphatase has been identified, but we do not know whether it can decalcify bone. Citric acid has been suggested.

Bone has been described as a hydroxyapatite, calcium phosphate. Ultramicroscopists find that it is deposited in minute ultramicroscopic crystallites which are loosely aggregated, so that each crystallite is more or less exposed to tissue fluids and can slip in or out of the crystalline state readily. Engstrom[7] has estimated that one gram of bone salt has an area of 130 square meters. Hendricks and Hill[8] earlier made an estimate of 180 square meters of surface exposure per gram.

It is thought that calcium, being an alkaline earth metal, is bound to carboxylate side chains of protein when the pH is in the region of 5 to 7 and proteins change from positive to negative charge, with consequent affinity for calcium. According to Cantarow and Schepartz,[6] the organic materials upon which bone salts are precipitated are proteins. These proteins are collagen (ossein), osseoalbuminoid, osseomucoid, and a mucoprotein probably containing chondroitinsulfuric acid as a prosthetic group. Certain enzymes have been identified, such as the phosphatases, peptidases, phosphorylases and proteinases. Ascorbic acid is essential in the production of intercellular substances of all connective and supporting tissue.

Kirkwood[9] of Yale states in the *Mechanism of Enzymes:*

> Proteins are amphoteric polymers of the highly polar amino-acids. The molecules undoubtedly possess characteristic distributions of electric charge which give rise to strong electrostatic interaction. Fluctuations induced by thermal motion may occur.

Bellucci and Wolff[4,5] probably produced fluctuations by thermal motion when cautery needle was applied to the bone in experimental cats and a microscopic picture similar to the "blue mantling" of otosclerosis was obtained.

Kirkwood[9] continues:

> When two protein molecules are separated by a distance R, the electric field of the fluctuating charge-distribution of each molecule alters the distribution of fluctuations in the charge and constellations of the mobile protons of the other in such a manner as to produce at the isoionic point a long range attractive force between them. . . . Crystalline proteins can exist in solution in varying degrees of aggregation. Subunits are bound together along closely fitting hydrophobic surfaces. Repulsion is greater than the cohesive forces of these highly charged units.

The microscopic picture which we see in otosclerosis is highly suggestive of an unstable electrochemical process which can fluctuate from one state to another. The etiology of this disease must now be attacked by the biochemist. The ultramicroscopist may add some information in the future. Perhaps the problem could be investigated from tissue cultures of the diseased bone, now that it is possible to collect specimens of this tissue. It seems conclusive that descriptive microscopic pathology has probed as far as it can in this disease. We are really dealing with a problem in crystallography. Some degradative en-

zyme must be the decalcifying substance which initiates the pathologic change. It is probably not the same enzyme in all cases. The problem is to identify it and counteract it locally. This is the attack initiated by those trying chelating agents. We suggest that such trials be made on animals whose stapedes have been experimentally ankylosed.

REFERENCES

1. Altmann, Franz, and Waltner, Jules G.: Slight operative injuries of the stapes; histopathologic study of a case, Arch. Otolaryng., 42:42-46, 1945.

2. Basek, Milos, and Fowler, Edmund P.: Anatomical factors in stapes-mobilization operations, Arch. Otolaryng., 63:589-597, 1956.

3. Bellucci, Richard J., and Wolff, Dorothy: Repair and consequences of surgical trauma to the ossicles and oval window of experimental animals, Ann. Otol. Rhin. Laryng., 67:400-429, 1958.

4. ———: Experimental induced ankylosis of the stapes, Laryngoscope, 69:229-240 (March) 1959.

5. ———: Regression of hearing improvement after mobilization of stapes, Laryngoscope, 69:241-247 (March) 1959.

6. Cantarow, Abraham, and Schepartz, Bernard: Calcium and Phosphorus Metabolism. In Biochemistry, Philadelphia, Saunders, 1954, pp. 609-621.

7. Engstrom, A.: Structure of the Bone from the Anatomical to the Molecular Level. In Bone Structure and Metabolism, Ciba Foundation Symposium, Boston, Little, Brown & Co., 1956, p. 8.

8. Hendricks, S. B., and Hill, W. L.: The nature of bone and phosphate rock, Proc. Nat. Acad. Sci., 36:731-737, 1950.

9. Kirkwood, John G.: The Nature of Forces Between Protein Molecules in Solution. In A Symposium on The Mechanism of Enzyme Action; edited by William D. McElroy and Bentley Glass, Baltimore, Johns Hopkins Press, 1954, pp. 4-23.

10. Kleiner, Israel Simon: Human Biochemistry, ed. 4, St. Louis, C. V. Mosby, 1954.

11. Lacroix, P.: The Histological Remodeling of Adult Bone. In Bone Structure and Metabolism, Ciba Foundation Symposium, Boston, Little, Brown & Co., 1956.

12. Lempert, Julius, and Wolff, Dorothy: Histopathology of the incus and the head of the malleus in cases of stapedial ankylosis, Arch. Otolaryng., 42:339-367, 1945.

13. Nager, George T., and Nager, M.: The arteries of the human middle ear, with particular regard to the blood supply of the auditory ossicles, Ann. Otol. Rhin. Laryng., 62:923-949 (Dec.) 1953.

14. Stein, Irvin; Stein, Raymond O., and Beller, Martin L.: Living Bone in Health and Disease, Philadelphia, Lippincott, 1955.

15. Wolff, Dorothy: Symposium. Histology and pathogenesis of otosclerosis. Otosclerosis, hypothesis of its origin and progress. Tr. Am. Acad. Ophth. & Otol. 54:11-22 (Nov., Suppl.) 1950.

II.

OTOSCLEROSIS WITH BILATERAL STAPES MOBILIZATION

FRANZ ALTMANN, M.D.
MILOS BASEK, M.D.
NEW YORK, NEW YORK
and
JACK V. D. HOUGH, M.D.
OKLAHOMA CITY, OKLAHOMA

WE have had the opportunity to examine histologically the temporal bones of a man, 52 years of age, in whom one of us (J.V.D.H.) had performed stapes mobilization on both the left and the right ear, four months and two weeks, respectively, before the patient's death from cerebral hemorrhage. The preoperative audiogram showed a bilateral mixed hearing loss. An audiogram taken three weeks after the first operation showed an average gain of 13 decibels for air conducted sounds. In the following months the patient's hearing in the surgically treated ear subjectively improved so much that he discarded his hearing aid. During the second operation, there was a quite satisfactory hearing improvement which continued postoperatively.

HISTOPATHOLOGIC FINDINGS

Left Ear (Stapes Mobilization Four Months Before Death)

Two small otosclerotic foci lie in the oval window region. One is situated at the posterior circumference of the window frame and is composed of more mature bone, without signs of activity. It had already extended on a broad front into the footplate before the operation. The other focus is situated at the anterosuperior quadrant of the bony window frame and consists mainly of more mature bone but shows some signs of activity. It has almost completely replaced the cartilage of the window frame. In a few places, fingerlike processes of newly formed "blue" bone (blue mantles) extend in vascular channels for a short distance into the adjacent capsule.

There has been substitution of the posterior and lower half of the middle third of the footplate by more immature otosclerotic bone, which also extends into the lower third of the posterior stapedial crus. Fracture through the bony bridge between posterior focus and footplate has been accomplished, and fragmentation of the otosclerotic portion of the footplate into several pieces has pushed some of the pieces slightly into the vestibule, but they are covered with a continuous endosteal membrane. Fracture extends through the otosclerotic portion of the posterior crus. Extensive new weblike bone has formed, uniting the fragments, including the posterior crus, with each other and with the window frame. Slight outward subluxation of the anterior part of the footplate with tear in the annular ligament filled with hyalinized tissue and fracture of the lowermost part of the anterior crus is evident, and there is apposition of a small amount of newly formed bone to

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Presented at the *Special Program* of the Otosclerosis Study Group and the Committee on Conservation of Hearing at the Sixty-Fourth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 11, 1959, Chicago.

the surface of the fragments. Areas of hyalinization and calcium deposits lie in the arterosuperior quadrant of the annular ligament.

There is an almost complete absence of reactive changes within the labyrinth.

It is surprising that in spite of the far advanced and still progressing reankylosis of the footplate, the hearing was still very good. However, it would undoubtedly have receded after the reankylosis had become complete.

Right Ear (Stapes Mobilization Thirteen Days Before Death)

An otosclerotic focus with very few signs of activity lies at the anterior, superior and inferior circumference of the window frame and is composed mainly of mature bone, in the vicinity of the footplate of more immature bone. Anteroinferior extension of the focus into the footplate has replaced almost the entire footplate by immature otosclerotic bone except for the posterosuperior marginal area, which consists of more mature bone. The bony bridge has been fractured through between the focus and the footplate, and the footplate has been fragmented into several pieces, some of which are pushed slightly into the vestibular lumen. Healed tears with calcareous deposits are present in the annular ligament and in the endosteum at the posterosuperior circumference of the footplate. Formation of new bone has begun around the fragments of the footplate and at the window frame in the area of the tear.

There is almost complete absence of reactive labyrinthine changes.

The speed with which the continuity of the endosteum was restored is remarkable. Although the footplate at the time of death was still mobile, new formation of bone had already started and, judging from the findings on the other side, would probably within a few months have produced reankylosis of the footplate.

COMMENT

The finer structure of the otosclerotic foci shows nothing remarkable. The only formations which should be mentioned here are the fingerlike processes of "blue" otosclerotic bone which, on the left side, extend in a few places from the surface of the otosclerotic focus into the adjacent labyrinthine capsule. They are typical *blue mantles*, a term first used by Manasse[7] and then more or less generally adopted by all authors. Blue mantles are areas of blue-staining bone which surround blood vessels in the labyrinthine capsule like a sheath. They are found at the periphery of otosclerotic foci or without connection with otosclerotic foci, particularly in the region of the semicircular canals. The structure of the bone of the blue mantles is identical with that composing the blue portion of the otosclerotic foci. When they are present, Weber points out the danger that these true "blue mantles" could be confused with the very thin membranes (also hematoxylin blue-staining) which normally cover the inner surface of the lacunae and the canaliculi of the bone as well as the walls of the vascular channels. These membranes are called *Grenzscheiden* in the German literature (*Broesike*,[3] *Schaffer*,[12] *Weidenreich*,[14] and others).

Unfortunately, the confusion predicted by Weber has actually occurred, particularly in the American literature. This is, for instance, evident from the following quotation from Lempert and Wolff[5]:

> Blue mantles are the walls of haversian canals which take an intensively blue dye with the hematoxylin. Blue mantles may occur in bone that is not otosclerotic, but they are always conspicuous in bone that is from patients with otosclerosis.

Nylen,[11] who uses the term "blue mantles" correctly, reports that Lempert and Wolff had observed changes of the blue-mantle type in the malleus and incus of operated cases of otosclerosis but completely overlooks the fact that "blue mantles" described by the two authors are not true blue mantles but simply *Grenzscheiden,* which represent a normal finding and have nothing to do with otosclerosis.

In a recent paper, Bellucci and Wolff[2] describe changes in the promontory of the cat after electrocoagulation of the oval window region. These show a striking similarity to the true blue mantles seen in otosclerosis. However, the interpretation given to the changes still adds to the existing confusion.

> A "blush" of purplish-red dye surrounds some of the vascular channels, while others remain normal in appearance. In these pathological areas decalcification has either already occurred or seems imminent. Clear areas along the border of the lesion suggest halisteresis. In these regions several osteocytes, recently freed from their lacunae, appear to congregate and form osteoclasts . . . other freed osteocytes become converted into fibroblasts filling up the space between vessel walls and bony walls. When the decalcification process has gone "far enough", i.e., presumably to supply physiological local demands, osteocytes will then be converted to osteoblasts and start regenerating bone.

This interpretation closely follows Wittmaack's concept[15] which, in view of the results of the investigations of O. Mayer,[8,9] Nager and Meyer,[10] Weber[13] and Greifenstein,[4] and others, has been discarded by practically all other authors.

It is now more or less generally agreed that a correct interpretation of the structure of otosclerotic bone or any other bone is impossible without a study of the fibrils, which is best done in polarized light, with silver impregnation or special stains such as Gram-Weigert (Weidenreich[14]). It then becomes evident beyond doubt that the blue mantles are composed of newly formed bone in spaces created by preceding destruction of the old bone of the capsule.

The findings in the two temporal bones just described are in good agreement with the observations of Lindsay[6] in two temporal bones. In a case reported by us previously,[1] fifteen months after what amounted to a fenestration of the uninvolved part of the footplate rather than an actual mobilization, an extensive and mostly inactive focus in the window frame had grown into the marginal area of the footplate but had left the main portion of the footplate intact. Practically no reactive changes were found around the broken pieces of the footplate, one of which was pushed toward the lumen of the vestibule, or on the surface of the otosclerotic focus with the exception of a few areas where somewhat greater activity was noted. The defect created in the footplate during operation was closed with a connective tissue layer; reactive labyrinthine changes were absent.

The almost complete absence of postoperative reactive changes around the otosclerotic foci and the fragments of the footplate could be explained by the fact that the otosclerotic focus itself was not fractured and that the broken pieces consisted of normal skeinlike bone which, in man, shows very little tendency to bony callus formation and, furthermore, that the annular ligament had not been torn. The findings in Lindsay's cases and in our last case seem to indicate that healed tears in the annular ligament are areas of predilection for new bone formation, a fact that should always be remembered during mobilization operations.

The findings in the six temporal bones so far reported and examined thirteen days to fifteen months after mobilization show quite clearly that procedures which

cause fractures through otosclerotic areas, fragmentation of otosclerotic portions of the footplate or extensive tears in the annular ligament will in a large percentage of cases be followed by reactive new formation of bone, which eventually leads to reankylosis of the mobilized stapes. The reactive new formation of bone seems to depend just as much on the intensity of the operative trauma or postoperative infection as on the degree of activity of the otosclerotic bone involved in the operation. Interventions on the footplate are necessarily more traumatic than indirect mobilization procedures, but even the latter, when carried out too energetically and causing extensive tears in the annular ligament, might stimulate reactive osteogenesis not only in the focus but also in the injured areas of the ligament.

The impressive results achieved with removal of the stapes, closure of the oval window with a graft and reconstitution of the ossicular chain seem to point the way to a far more direct approach to the treatment of otosclerotic hearing loss than through the methods of stapedolysis. However, it remains to be seen whether the otosclerotic bone at the window frame, "activated" by the operative trauma, will not in many instances grow into the tissue graft and again produce eventually, although much later, an ankylosis of the new footplate.

The practically complete absence of more serious labyrinthine reactions in the histologically examined cases and the closure of a hole in the footplate with a connective tissue membrane in one observation indicate that the labyrinth has a considerable and unexpected resistance to trauma. However, a word of caution seems appropriate in this respect. Interventions at the oval window, even when carried out with the greatest caution, are potentially dangerous and only time will tell whether their obvious advantages really outweigh their inherent dangers.

REFERENCES

1. Altmann, Franz, and Basek, Milos: Histological examination of a case of otosclerosis fifteen months after stapes-mobilization operation, Arch. Otolaryng., 68:314-324 (Sept.) 1958.
2. Bellucci, Richard J., and Wolff, Dorothy: Experimentally induced ankylosis of the stapes, Laryngoscope, 69:240-241 (March) 1959.
3. Broesike, G.: Uber die sogenannten Grenzscheiden des Knochencanalsystems, nebst Bemerkungen über die Keratinsubstanzen, Arch. f. mikr. Anat., 26:88-125, 1885.
4. Greifenstein, A.: Vergleichende Untersuchungen zur Histologie der Otosklerose, Arch. Ohren-, Nasen-, u. Kehlkopfh., 139:14-98, 1935.
5. Lempert, Julius, and Wolff, Dorothy: Otosclerosis: theory of its origin and development, Arch. Otolaryng., 50:115-155 (Aug.) 1949.
6. Lindsay, John R., et al.: Histopathologic changes following fenestration and stapes mobilization, Tr. Am. Acad. Ophth. & Otol., 63:187-205, 1959.
7. Manasse, P.: Neue Untersuchungen zur Otosklerosefrage, Ztschr. f. Ohrenh., 82: 76-96, 1922.
8. Mayer, O.: Untersuchungen über die Otosklerose, Vienna, Hölder, 1917.
9. ———: Referat uber die pathologische Anatomie und Pathogenese der Otosklerose, Trans. II. Internat. Congress of Otolaryngology, Madrid, 1932. Imp. Blass.
10. Nager, F. R., and Meyer, M.: Die Erkrankungen des Knochensystems und ihre Erscheinungen an der Innenohrkapsel des Menschen, Berlin, S. Karger, 1932.
11. Nylen, B.: Histopathological investigations on the localization, number, activity and extent of Otosclerotic foci, Upsala läkaref. förh., Ny Foljd, 54:1,1949.
12. Schaffer, Joseph: Lehrbuch der Histologie und Histogenese, ed. 3, Berlin & Wien, Urban & Schwarzenberg, 1933.
13. Weber, M.: Otosklerose und Umbau der Labyrinthkapsel, Leipzig, Offizin Poeschel & Trepte, 1935.
14. Weidenreich, F.: Das Knochengewebe. Handbuch der mikroskopischen Anatomie des Menschen, ed. by Moellendorff, Berlin, J. Springer, 1930, vol. II, Pt. 2, p. 391.
15. Wittmaack, Karl: Die Otosklerose auf Grund eigener Forschungen, Jena, G. Fischer, 1919.

In Memoriam

Robert Elmer Anslow.....Detroit, Michigan
March 16, 1960

Edgar B. Burchell.....Larchmont, New York

Eugene L. Christensen.....Los Angeles, California
March 22, 1960

Frank L. Cohen.....Philadelphia, Pennsylvania
April 18, 1960

Daniel S. Cunning.....New York, New York
March 25, 1960

Herbert H. Glosser.....Wellsville, New York
March 11, 1960

Albert Hiram Herr.....Lima, Ohio
April 15, 1960

Donald MacLean Howell.....Saginaw, Michigan
April 2, 1960

Samuel I. Kaufman.....Chicago, Illinois
April 22, 1960

George A. Leahey.....Lowell, Massachusetts
April 1, 1960

James H. Maxwell.....Ann Arbor, Michigan
June 2, 1960

Emil R. Mayerberg.....Wilmington, Delaware
May 8, 1960

James W. McKennan.....Washington, Pennsylvania
March 7, 1960

William Wallace Morrison.....New York, New York
April 17, 1960

Charles F. Nelson.....Cleveland, Ohio
March 17, 1960

Robert E. Parrish.....San Antonio, Texas
May 29, 1960

Ellis H. Steffensen.....Detroit, Michigan
April 9, 1960

William P. Woodworth.....Detroit, Michigan
May 22, 1960

TRANSACTIONS

American Academy of Ophthalmology and Otolaryngology

Official Publication of the American Academy of Ophthalmology and Otolaryngology

Published under the direction of the Editorial Board
WILLIAM L. BENEDICT, Rochester, Minnesota, *Editor*
W. HOWARD MORRISON, Omaha, Nebraska, *Associate Editor*
HENRY L. WILLIAMS, Rochester, Minnesota, *Associate Editor*
Printed by Douglas Printing Company, 109 North 18th Street, Omaha 2, Nebraska

William L. Benedict, M.D., LL.D., Executive Secretary-Treasurer
American Academy of Ophthalmology and Otolaryngology
15 Second Street S.W., Rochester, Minnesota

THE SIXTY-FIFTH ANNUAL MEETING

The Sixty-Fifth Annual Meeting of the Academy will take place on October 8 to 14 in the Palmer House. The Scientific Program, the Scientific Exhibit, the Instruction Courses, and the commercial exhibits will, as in the past few years, constitute the major activities of the convention. An exceptionally interesting array of scientific contributions, papers, motion pictures, forums and case reports has been scheduled by the Program Committee.

The commercial exhibits have again spread to the seventh floor, where half the members spend half their time each day. These exhibits are in rooms instead of booths, so the close attention of the prospective buyer is not distracted by heavy traffic found on the fourth floor. Be sure to look up the exhibitors on the seventh floor.

The scientific exhibit on the Club Floor above the Exhibition Hall will cover new subjects. Every member will do well to schedule definite time for a complete study of the scientific demonstrations.

The Teachers' Sections will hold separate meetings on Sunday morning beginning at ten o'clock: Ophthalmologists in the Grand Ballroom; Otolaryngologists in the Crystal Room.

The annual banquet will be held in the Grand Ballroom on Monday evening. There will be no speeches but there will be the grandest floor show in Chicago. The special evening entertainment has been arranged by Ralph Ginsburgh, who has supplied magnificent entertainment at our last few banquets. Tables may be reserved through purchase of tickets in the registration area. Dress is optional.

Minor changes in registration will facilitate movement in the early hours of the convention. Advance registration has been provided to a greater number of nonmembers and to exhibitors. See the announcement in the Official Notices in the Program Section of this TRANSACTIONS.

The Program Committee has noted a tendency to diminishing attendance on Thursday and Friday in instruction courses as well as in the scientific assembly. Many members arrive in Chicago several days prior to the Academy week for meetings of special instruction courses given by institutions or individuals in Chicago and for scientific ses-

sions of groups, some of which are Academy affiliates. It is the opinion of the Program Committee that the meetings immediately preceding the opening of the Academy convention seriously curtail the attendance at the regular sessions of the Academy. The Committee has offered all assistance possible in aiding such groups to arrange their meetings on Friday and Saturday following the Academy sessions. A more fully integrated program of papers will give prominence to special subjects in the broad field of sensory diseases and will keep the Academy activities unified.

FINANCIAL REPORT

Annual Audit for Calendar Year 1959

May 5, 1960

American Academy of
Ophthalmology and Otolaryngology
Rochester, Minnesota

Gentlemen:

We have examined the balance sheet of the American Academy of Ophthalmology and Otolaryngology (an unincorporated association), as of December 31, 1959, and the related statement of income and net worth for the year then ended. Our examination was made in accordance with generally accepted auditing standards and accordingly included such tests of the accounting records and such other auditing procedures as we considered necessary in the circumstances.

We submit herewith our report which includes the following financial statements:

Exhibit

I Balance Sheet as of December 31, 1959
II Statement of Income and Net Worth for the year ended December 31, 1959
III Analysis of Grants-in-Aid and Special Contributions for the year ended December 31, 1959
IV Securities Owned, December 31, 1959

We were unable to satisfy ourselves that the inventory value of the Atlas of Otolaryngic Pathology ($22,768.96), as shown on the accompanying balance sheet (Exhibit I), represents net realizeable value. Sales of this Atlas have decreased steadily the past two years. The Academy has recovered approximately half of its investment in this Atlas, and if the sales trend continues to decrease, it appears doubtful that the Academy will recover its remaining investment.

The Atlas of Slit Lamp Gonioscopy is another proposed publication being sponsored by the Academy. It has been in process of compilation since 1956. To date the Academy has invested $15,885.35 towards development of this Atlas, which amount is shown under "other assets" on the accompanying balance sheet (Exhibit I). No progress was made towards completion during 1959, and we were unable to determine satisfactorily whether or not the Academy will ever recover the costs incurred to date.

In our opinion, subject to the comments in the preceding paragraphs relating to possible revaluation of the atlases, the accompanying balance sheet and related statement of income and net worth present fairly the financial position of the American Academy of Ophthalmology and Otolaryngology as of December 31, 1959, and the results of its operations for the year then ended, in conformity with generally accepted accounting principles applied on a basis consistent with that of the preceding year.

Comments on operations and the scope of our examination are included in the following paragraphs of this report.

HISTORY AND ORGANIZATION

The American Academy of Ophthalmology and Otolaryngology with office in Rochester, Minnesota, was organized in 1896. The purpose of the Academy is to promote and advance the science and art of medicine appertaining to the eye, ear, nose and throat.

The Academy's insurance coverage at December 31, 1959, was as follows:

Type of Coverage	*Amount*
Fire and extended coverage:	
Contents (80% co-insurance)	$ 10,000
Bound volumes, manuals and abstracts	15,000
Equipment floater	25,157
General liability:	
Bodily injury	100,000/300,000
Property damage	5,000/25,000
Forgery and check alteration	10,000

In addition the Academy carried workmen's compensation insurance at statutory amounts including coverage for help hired at the annual convention; a fidelity bond on the executive secretary in the amount of $25,000.00 and other office employees, $2,500.00. A broad form policy is being carried covering the destruction or disappearance of money, securities and other property up to $3,225.00 within the premises, and $2,100.00 outside the premises when conveyed by a messenger. At the annual convention, this coverage is increased to $40,000.00 with a limit of $20,000.00 on money.

FINANCIAL POSITION

Cash in banks and on hand at December 31, 1959, consisted of the following balances (in rounded amounts):

On hand:	
Petty cash	$ 25.00
In banks:	
Northwestern National Bank, Rochester, Minnesota	56,910.00
First National Bank, Minneapolis	6,195.00
Bank of Montreal, Toronto, Canada	1,670.00
	$64,800.00

Accounts receivable at December 31, 1959, increased approximately $7,000.00 over the balance at December 31, 1958. This increase was in amounts due from advertisers in the TRANSACTIONS and from testing of Tonometers. Approximately 94% of the total of "Accounts receivable—Other" outstanding at December 31, 1959, represent current billings.

The inventory at December 31, 1959, primarily represents books and publications held for sale to members and others. Remarks pertaining to the inventory valuations as represented by the Atlas of Otolaryngic Pathology are set forth in the preceding pages of this report.

The investment in stocks and bonds totaling $423,303.60, is represented by the securities set forth in Exhibit IV. The market value of these securities at December 31, 1959, was approximately $68,000.00 in excess of cost.

During the annual meeting of the Academy held October 15, 1959, a resolution was adopted authorizing the creation of an "Educational Trust of the American Academy of Ophthalmology and Otolaryngology." The Council has also authorized the Executive Secretary-Treasurer to transfer to the Educational Trust the securities in the "Educational Fund," substantially as shown in Exhibit IV of this report, and now held in custodianship by the Trust Department of the First National Bank, Minneapolis, Minnesota. Although final details on the "Trust" arrangement had not been completed at the date of this report, the effect of the creation of this trust by the Academy is to restrict the use of these funds to purposes as set forth in the proposed trust agreement.

Except for equipment acquired prior to September 1, 1942, which was appraised at $1,420.28 as of January 1, 1944, furniture and fixtures are stated at cost. New equipment additions were made during the current year in the amount of approximately $1,700.00.

The cost of motion picture films and slides entitled "Embryology of the Eye," totaling $41,775.00, and "Embryology of the Ear," totaling $104,516.62, represents the original production cost of these films and slides prepared by Sturgis-Grant Productions, Inc., and are being amortized at a rate that appears reasonable.

OPERATIONS

A comparative statement of income for the years ended December 31, 1959 and 1958, in condensed form, is as follows: [See next page.]

The decrease in net income of $22,950 from a year ago, resulted, in part, from a decrease of $12,500 in income from TRANSACTIONS (revenues less expenses) and in part from increased scholarship awards of $6,800 and general increases in other expenses.

The increase in market value of securities owned at December 31, 1959, over December 31, 1958, is set forth below: [See next page.]

The Academy follows the policy of reflecting the fluctuation in market values of securities in its net worth.

	Year ended: Dec. 31, 1959	Dec. 31, 1958	Increase Decrease
Revenues	$304,000	$280,500	$ 23,500
Operating Expenses:			
General administrative	116,900	108,900	8,000
Educational Fund	25,500	18,000	7,500
Home Study Courses	12,500	10,200	2,300
Convention expenses	63,900	57,200	6,700
TRANSACTIONS	90,300	68,500	21,800
Total operating expenses	309,100	262,800	46,300
Operating income **(loss)**	**5,100**	17,700	**22,800**
Other Income **(deductions)**	**100**	50	**150**
Net income **(loss)**	**$ 5,200**	$ 17,750	**$22,950**

	COST	MARKET	INCREASE
December 31, 1959 (Exhibit IV)	$355,191.54	$423,303.60	$ 68,112.06
December 31, 1958 (Audit report)	302,402.68	367,172.28	64,769.60
Difference	$ 52,788.86	$ 56,131.32	$ 3,342.46

SCOPE OF AUDIT

Our examination included, but was not limited to, the following procedures:

Cash in banks was reconciled with the balances confirmed to us by the depositaries.

Accounts receivable balances as shown on the individual subsidiary records were reconciled with the control accounts as of December 31, 1959. We circularized individual balances (except membership dues receivable) on a test basis as of December 31, 1959, and in the replies reviewed to date representing approximately 41% of accounts receivable (other than membership dues), no material differences were disclosed. We reviewed the collectibility of the accounts receivable and no material losses or adjustments are anticipated in the collection of the accounts outstanding at December 31, 1959.

Inventories were determined by physical count taken by Academy employees as of December 31, 1959. We observed the inventory taking and made test counts of quantities. Our examination included the testing of inventory prices, extensions, and footings. This review indicated that quantities were accurately determined and that the inventory was priced at cost.

Prepaid insurance and postage were determined to be properly applicable to future operations.

Investments in stocks and bonds are held for safekeeping by the Trust Department of the First National Bank of Minneapolis, Minnesota. We confirmed the existence of the securities directly with the custodian.

Fixed asset accounts were reviewed and additions were traced to invoices and other supporting data. We checked the depreciation computation and reviewed the depreciation rates which appeared reasonable in relation to the estimated life of the depreciable property.

We made test checks of the records of accrued expenses, accrued payroll taxes and other liability accounts. We analyzed the grant-in-aid accounts for the year ended December 31, 1959, comparing receipts with "notification of grant-in-aid awards" received from the National Institutes of Health, Department of Health, Education and Welfare, Bethesda, Maryland. Expenditures from grant-in-aid funds were checked on a test basis against invoices and other supporting data.

Respectfully submitted,
BYERS, WOBSCHALL & MILLER
Certified Public Accountants
By Edw. A. Wobschall, C.P.A.

EXHIBIT I

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
(*An Unincorporated Association*)
Balance Sheet as at December 31, 1959

ASSETS

Current Assets:			
Cash on hand and in banks			$ 64,801.74
Membership dues receivable—delinquent			1,100.00
Accounts receivable—other			8,507.84
Inventories, at cost:			
Abstracts, manuals, films		$ 27,213.93	
Atlas of Otolaryngic Pathology		22,768.96	49,982.89
Prepaid insurance			423.00
Prepaid postage, etc.			70.70
Total current assets			124,886.17
Investments, at market value (Exhibit IV): (*see note*)			
Bonds and stocks (cost $355,191.54)			423,303.60
Other Assets:			
Atlas of Slit Lamp Gonioscopy (cost of Atlas in process of compilation)			15,885.35

Fixed Assets, at cost:	GROSS	ACCUMULATED DEPRECIATION	NET	
Furniture and equipment	$ 21,182.12	$ 11,992.59	$ 9,189.53	
Films and slides:				
Embryology of the Eye	41,775.00	40,731.25	1,043.75	
Embryology of the Ear	104,516.62	49,263.42	55,253.20	
	$167,473.74	$101,987.26	$ 65,486.48	$ 65,486.48
				$629,561.60

LIABILITIES AND NET WORTH

Current Liabilities:	
Accrued expenses	$ 12,356.17
Due to audiometer testing stations	2,400.00
Accrued payroll taxes	1,044.15
Prepayments for Bound Volumes, etc.	5,260.00
Grants-in-aid (unexpended balances)	111,456.59
Deferred income, membership dues	150.00
Total current liabilities	132,666.91
Wherry Memorial Lecture Fund Balance	6,072.05
Net Worth (Exhibit II)—see note	490,822.64
	$629,561.60

(The accompanying note in Exhibit I-A is an integral part of this balance sheet.)

EXHIBIT I-A

NOTE TO BALANCE SHEET

The portion of net worth at December 31, 1959, representing the proposed "Educational Trust" created by action of the Academy during its annual meeting on October 15, 1959, has not been segregated from the total net worth inasmuch as execution of the "trust agreement" had not been completed. However, securities listed under "Educational Fund" on Exhibit IV are substantially similar to those outstanding at October 15, 1959, and, therefore, this action of the Academy imposes a restriction on the use of net worth at December 31, 1959, for purposes set forth in the "Educational Trust Agreement" in the amount of approximately $417,000.00.

EXHIBIT II

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Statement of Income and Net Worth
For the Year Ended December 31, 1959

Revenues:			
Membership dues			$ 74,615.00
Membership applications			4,446.00
Abstracts, manuals and monographs			13,198.79
Certificates			1,260.50
Films and slides			1,700.00
Testing and calibration of tonometers			1,232.00
Royalties			599.40
Convention			93,128.00
Home Study Courses			9,360.00
TRANSACTIONS			85,654.58
Income from securities:			
Interest		$ 3,287.77	
Dividends		15,481.38	18,769.15
Other			30.00
Total revenues			303,993.42
Operating Expenses:			
General administrative		116,924.94	
Educational Fund:			
Trustee fees	$ 378.70		
Depreciation, films and slides	14,629.16		
Embryology of Ear, Album	126.48		
Scholarship awards	8,600.00		
Supplements	1,781.85	25,516.19	
Home Study Courses		12,450.07	
Convention expenses		63,927.13	
TRANSACTIONS		90,303.99	309,122.32
Operating loss			**5,128.90**
Loss on Sale of Securities			84.63
Net loss for the year			**5,213.53**
Net Worth, December 31, 1958			492,693.71
			487,480.18
Add—Increase in market value of securities during 1959			3,342.46
Net Worth, December 31, 1959			$490,822.64

EXHIBIT III

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Analysis of Grants-in-Aid and Special Contributions for the Year Ended December 31, 1959

			UNEXPENDED BALANCE DEC. 31, 1959
N. I. H. GRANT-IN-AID NO. 3-B-9023 (c1) & 3-B-9023 (c2) (UVEITIS)	3-B-9023 (c1)	3-B-9023 (c2)	
Balance, December 31, 1958	$23,378.20		
Receipts for year 1959:			
Treasury of United States	15,813.98	$43,056.00	
	39,192.18	43,056.00	
Disbursements during 1959	22,302.11	9,598.15	
Balance, December 31, 1959	$16,890.07	$33,457.85	$50,347.92
N. I. H. GRANT-IN-AID NO. 3-B-9045 (c1) & B-2375 (c2) (HEARING)	3-B-9045 (c1)	B-2375 (c2)	
Balance, December 31, 1958	$16,416.24		
Receipts for year 1959:			
Treasury of United States	29,618.30	$58,891.25	
Transfers by order of N. I. H.	**8,308.75**	8,308.75	
	37,725.79	67,200.00	
Disbursements during 1959	37,725.79	9,492.87	
Balance, December 31, 1959	None	$57,707.13	57,707.13
N. I. H. GRANT-IN-AID NO. 2-B-5926 (EMPLOYMENT REGISTRY FOR SCIENTISTS)			
Balance, December 31, 1958		None	
Receipts for year 1959:			
Treasury of United States		$ 3,938.00	
		3,938.00	
Disbursements during 1959		536.46	
Balance, December 31, 1959		$ 3,401.54	3,401.54
SUB-COMMITTEE ON NOISE IN INDUSTRY			
Balance, December 31, 1958		None	
Receipts, for year 1959 (contributions)		$37,620.37	
		37,620.37	
Disbursements during 1959:			
Payroll of Sub-committee	$33,335.60		
Social security tax on above	667.18		
Transfers to Sub-committee—other	3,617.59	37,620.37	
Balance, December 31, 1959		None	
Total unexpended balance, December 31, 1959			$111,456.59
WHERRY MEMORIAL LECTURE FUND			
Balance, December 31, 1958			$ 6,156.05
Receipts for 1959—Dividends			216.00
			6,372.05
Disbursements during 1959—Expenses at 1959 convention			300.00
Balance, December 31, 1959			$ 6,072.05

EXHIBIT IV

American Academy of Ophthalmology and Otolaryngology
Securities Owned, December 31, 1959

EDUCATIONAL FUND

BONDS	RATE %	MATURITY	PAR VALUE	COST	MARKET VALUE	
Citizens Building Co. of Cleveland, 1st Leasehold Sinking Fund	6	1 /1/46	$ 5,000.00	.....	.....	(1)
Van Sweringen Co., Certificates of Indebtedness, Series 1925	6	12/31/48	790.70	$ 822.03	$ 15.81	(2)
Dominion of Canada	3¼	10/ 1/79	3,000.00	2,595.00	2,359.50	(3)
Dominion of Canada	3¼	10/ 1/79	5,500.00	5,612.34	4,325.75	(3)
Dominion of Canada	4½	9/ 1/83	9,500.00	9,500.00	8,573.75	(3)
Dominion of Canada	4½	9/ 1/83	4,000.00	3,750.00	3,610.00	(3)
U. S. Treasury Bonds	3¼	5/15/85	40,000.00	39,350.00	33,650.00	
U. S. Treasury Notes, Series B	3¼	5/15/60	20,000.00	19,912.50	19,862.50	
U. S. Treasury Notes, Series B	5	8/15/64	20,000.00	20,000.00	20,137.50	
American Telephone & Telegraph Debentures	5⅜	11/ 1/86	10,000.00	10,225.00	10,500.00	
Total Bonds				111,766.87	103,034.81	

STOCKS	KIND	PAR VALUE	SHARES			
Citizens Building Co. of Cleveland.	Shares	No par	50	.....	.....	(1)
Commonwealth Edison Co.	Common	$ 25.00	104	2,286.21	5,967.00	
E. I. du Pont de Nemours & Co. ..	Common	5.00	60	2,347.31	15,840.00	
Northern Illinois Gas Co.	Common	5.00	34	401.44	1,037.00	
Investors Diversified Services	Common A	1.00	100	6,785.95	23,400.00	
Bank of America Nat'l Trust & Savings Assn.	Common	6.25	100	3,950.00	4,925.00	
Weco Products	Common	1.00	300	4,125.00	4,650.00	
Farwell, Ozmun Kirk & Co.	Capital	50.00	100	7,800.00	7,500.00	
Louisville Gas & Electric Co.	Preferred—5%	25.00	200	5,450.00	5,500.00	
Kaiser Aluminum & Chemical	Preferred—4¼%	100.00	50	5,000.00	6,050.00	
Texas Eastern Transmission	Preferred—5.75	100.00	150	15,038.00	17,025.00	
The One William Street Fund, Inc..	Capital	1.00	2,040	25,010.40	22,419.60	
Agricultural Insurance Co.	Capital	10.00	110	4,075.00	3,080.00	
Boston Fund	Common	1.00	600	11,634.00	10,644.00	
Minneapolis Gas Co.	Preferred—5.50	100.00	20	2,060.00	1,960.00	
Incorporated Investors	Capital	1.00	4,021	34,530.70	40,531.68	
Massachusetts Investors Trust	Trust Certif. B/1	.33⅓	4,037	21,329.29	57,042.81	
Incorporated Income Fund	Capital	1.00	3,272	32,617.80	30,102.40	
Wellington Fund, Inc.	Common	1.00	4,042	53,583.57	57,194.30	
Total Stocks				238,024.67	314,868.79	
TOTAL SECURITIES				349,791.54	417,903.60	

WHERRY MEMORIAL FUND

Rochester Bldg. & Loan Assn.	Capital	100.00	5,400.00	5,400.00	
			$355,191.54	$423,303.60	

(1) Charged off in 1955—no market available

(2) In process of liquidation (original par value, 6,000.00)

(3) In U. S. dollars

SIXTY-FIFTH ANNUAL MEETING

American Academy of Ophthalmology and Otolaryngology

OCTOBER 9 TO 14, 1960

PALMER HOUSE

Chicago, Illinois

OFFICIAL NOTICES

Convention Call

Under the authority of the Constitution and with the approval of the Council, I herewith proclaim that the Sixty-Fifth Annual Session of the American Academy of Ophthalmology and Otolaryngology will convene at the Palmer House, Chicago, Illinois, on Monday, October 10, 1960, at 9:30 a.m., and will continue through Friday, October 14, 1960.

DEAN M. LIERLE, M.D.
President

Attest:
WILLIAM L. BENEDICT, M.D.
Executive Secretary-Treasurer

Council Meetings

The Council of the American Academy of Ophthalmology and Otolaryngology will convene at nine o'clock, Saturday morning, October 8, 1960, in Private Dining Room 4 of the Palmer House, Chicago. There will also be a meeting of the Council on Wednesday, October 12, at 12:45 p.m. in Private Dining Room 5. Members desiring to be heard or to present business will confer with the Executive Secretary-Treasurer for appointments.

DEAN M. LIERLE, M.D.
President

Business Meeting

The Annual Business Meeting of the Academy will be held in Private Dining Room 18 of the Palmer House on Thursday, October 13, 1960, at 5:30 p.m.

The Order of Business:

1. Call to order
2. Report of Executive Secretary-Treasurer
3. Report of the Senior Member of the Council
4. Election of Fellows
5. Election of Officers
6. Unfinished business
7. New business

All Fellows and candidates for election to Fellowship are urged to attend.

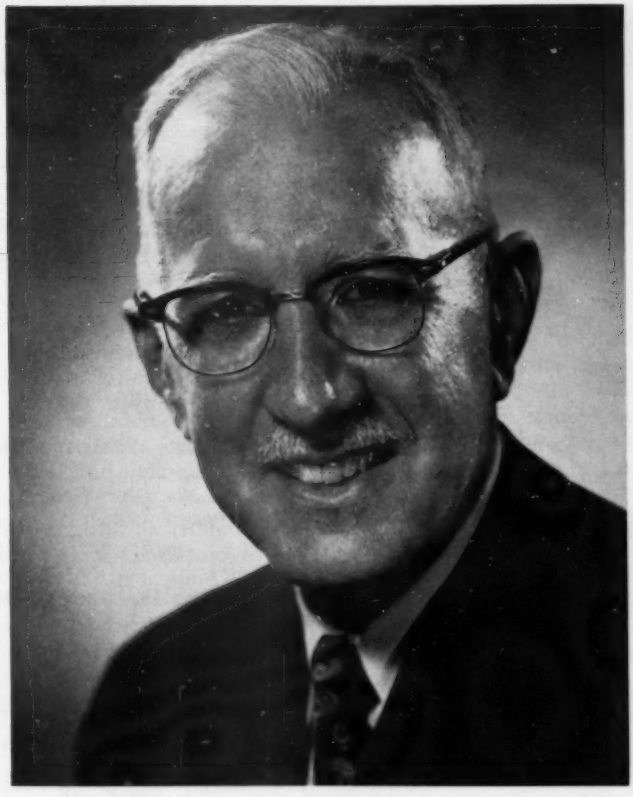
New Business

Proposals for new activities often are presented too late during the Academy session to afford time for deliberation. Therefore, new items requiring Council

President 1960

IOWA CITY, IOWA

Guest of Honor



GORDON D. HOOPLE, M.D.

MEDICAL DIRECTOR OF SYRACUSE UNIVERSITY
HEARING AND SPEECH CENTER

Bachelor of Science, Syracuse University, 1915; Doctor of Medicine, Syracuse University, 1919.

Life Fellow of the American Academy of Ophthalmology and Otolaryngology; Vice President, 1959; Member and Vice Chairman of the Committee on Conservation of Hearing, and Member of the Subcommittee on Noise; Member of the Armed Forces Committee.

Member of the American Otological Society, and President in 1952. Member of American Laryngological Association, and of American Broncho-Esophagological Society.

Trustee of Syracuse University.

action should be presented in writing to the Executive Secretary-Treasurer early so that the Committee concerned may have an opportunity to study the matter prior to presentation to the Council.

Public Relations

Contributors to the scientific program are requested to send copies of their papers to the Secretary for Public Relations, Glen G. Gibson, M.D., 2031 Locust Street, Philadelphia 3, at the earliest possible moment.

Exhibits

The Scientific and Technical Exhibits will open at nine o'clock on Sunday morning, October 9. They will close at four o'clock Sunday afternoon for the President's Reception. From Monday through Friday they will open at 8:30 a.m. The Scientific Exhibit will close at 5:30 p.m. each day, and the Technical Exhibit at 5:00 p.m., except that they will both close on Friday, October 14, at twelve o'clock noon.

Dues

Membership card for 1960 is required for registration at the Academy meeting. Those who have not paid dues for 1960 are requested to remit to W. L. Benedict, M.D., Executive Secretary-Treasurer, 15 Second Street S.W., Rochester, Minn. In case of loss, a duplicate membership card may be obtained upon request.

Registration

The badge secured at the time of registration is essential for admittance to all activities.

TIME AND PLACE

Sunday—9:00 a.m. to 4:00 p.m.

Monday through Thursday — 8:30 a.m. to 5:00 p.m.

Foyer to the Grand Ballroom on Fourth Floor of the Palmer House.

Each registrant is required to fill out a registration card and present it to the proper desk as outlined below. Registration cards will be found on tables in the center of the Foyer.

To facilitate registration, the following sections will be provided:

TO RIGHT AS YOU ENTER FOYER
(ADVANCE REGISTRATION)

Fellows who have ordered Instruction Courses in advance

Instructors

Contributors to Scientific Program

Contributors to Scientific Exhibit

Candidates for Academy Fellowship

TO LEFT AS YOU ENTER FOYER

All Fellows not in above classifications. Membership card for 1960 is required for registration. Dues outstanding may be paid at this desk.

Nonmembers (Fee of $10.00 required)
Guest physicians
Nonmedical guests

Nonmembers (No fee required)
Residents who hold Academy Guest Card for 1960 obtained by Head of Department

Special Guests (1960 Guest Card required)

Orthoptic Technicians (A.A.O.T.) Tickets for the Orthoptic Technicians' Instruction Courses may be purchased here.

Guests

Physicians who are not members are welcome to attend the meeting of the Academy as guests and to participate in all the activities. All guests, however, will be required to register, and to pay a fee of $10.00.

On written request by members of the Academy to the Executive Secretary in advance of the annual meeting, nurses, technicians, assistants, social workers and others will be admitted as guests without fee. Guest cards will be mailed to such persons in advance of the meeting. All other guests will pay a registration fee of $10.00. Requests for registration without fee made at the registration desk at the time of the meeting will not be honored unless the guest is accompanied by a member or the member has sent a letter with the guest.

Residents

Men in training in accepted residencies, or those now completing a full academic year (nine months) in basic study, may register without fee upon presentation of a guest card obtained by written request of their Department Heads.

Requests from Department Heads for guest cards should be sent to W. L. Benedict, M.D., 15 Second Street S.W., Rochester, Minn., by September 29.

Instruction Course Tickets

The tickets for courses in ophthalmology and the tickets for courses in otolaryngology and maxillofacial surgery will be sold at separate desks in the Registration area. Bulletin boards behind each desk will indicate what tickets are available. Please consult these boards in making your selections. Bear in mind that individual courses are for one hour only; continuous courses run for two or more hours. Price of tickets is $2.00 per hour.

It is necessary to register and to be wearing a regulation badge in order to purchase instruction course tickets.

Ladies' Registration

The wives of members and guests are urged to register at the Ladies' Headquarters on the eighth floor of the Palmer House, Room 832. Registration will open at nine o'clock, Sunday morning, October 9, and will close Wednesday evening, October 12.

CONVENTION ACTIVITIES

PRESIDENT'S RECEPTION

Grand Ballroom

Sunday, 4:30 p.m.

President Dean M. Lierle and Mrs. Lierle invite all members and guests of the Academy to a reception on Sunday, October 9, at 4:30 p.m. in the Grand Ballroom.

XVII JACKSON MEMORIAL LECTURE

Sponsored by the Ophthalmic Publishing Co.

Dr. Lorenz E. Zimmerman, of Washington, D. C., Chief of the Section on Ophthalmic Pathology, Armed Forces Institute of Pathology, Physician-Specialist in the Central Laboratory for Pathologic Anatomy and Research, Veterans Administration; and Consultant in Pathology to the American Academy of Ophthalmology and Otolaryngology, will deliver the XVII Jackson Memorial Lecture at the scientific session of the Section on Ophthalmology, Tuesday morning, October 11. The subject of his lecture will be "The Registry of Ophthalmic Pathology: Past, Present and Future."

XVI WHERRY MEMORIAL LECTURE

Sponsored by the Wherry Memorial Fund

The XVI Wherry Memorial Lecture will be delivered at the scientific session of the Section on Otolaryngology, Tuesday afternoon, October 11, by Dr. Anderson C. Hilding, Research Laboratory, St. Luke's Hospital, Duluth, Minnesota. His subject will be "Experimental Studies on Some Little Understood Aspects of the Physiology of the Respiratory Tract and Their Clinical Importance."

THE ANNUAL BANQUET

Grand Ballroom

Monday, 7:00 p.m.

Doors open at 6:30 p.m. Bar service will be available. Dress optional.

Tickets will be on sale beginning Sunday morning at nine o'clock in the Registration Area. Tables may be reserved, ten places to a table. Sale will close at two o'clock Monday afternoon.

FACULTY BUFFET LUNCHEON

Private Dining Room 14

Thursday, 12:30 p.m.

Members of the Instruction Courses Faculty, the Scientific Exhibitors, and authors and discussers on the Scientific Programs are invited to a Buffet Luncheon in Private Dining Room 14, Thursday at 12:30 p.m.

LADIES' PROGRAM

Wives and guests of members of the Academy are invited to participate in the special program of events planned for their pleasure and convenience by the Academy.

Coffee and rolls will be served daily from 10:30 to 11:30 a.m. and from 4:00 to 5:00 p.m. in the Ladies' Hospitality Suite, Rooms 832-834. On Sunday afternoon the coffee hour will be from three to four o'clock.

ALUMNI FUNCTIONS — 1960

The following alumni groups will hold dinners, luncheons, or cocktail parties during the Academy meeting. Most of these functions will be held on Wednesday evening, October 12, 1960. (A complete listing including time, place of function and address of the secretary for each group will appear in the Pocket Program issued at the Academy meeting.)

Brooklyn Eye and Ear Hospital Alumni

Dr. J. Mackenzie Brown Annual Dinner

Buffalo Alumni

College of Medical Evangelists—Ophthalmology and Otolaryngology

Cook County Hospital Ophthalmological and Otolaryngological Alumni

Duke Alumni

Henry Ford Hospital Alumni

Gill Memorial Eye, Ear and Throat Hospital Alumni—Annual Spring Congress

Grady Memorial Hospital Alumni

Harper Hospital Alumni

Heed Ophthalmic Foundation Alumni (Society of Heed Fellows)

Indiana Alumni and Staff

Johns Hopkins Hospital, Wilmer Institute Alumni

Dr. David H. Jones Alumni

Dr. Arlington C. Krause Luncheon

Dr. John R. Lindsay Alumni

Manhattan Eye, Ear & Throat Hospital Alumni

Dr. William A. Mann Ophthalmological Alumni of V. A. Hines

Massachusetts Eye and Ear Infirmary & Harvard Medical School Alumni

Mayo Clinic Alumni

Michael Reese Hospital Eye Alumni

Mount Sinai Hospital of New York—Otolaryngology Alumni

Newark Eye and Ear Infirmary Alumni

New York Eye and Ear Infirmary Alumni Association

New York Hospital Eye Alumni

New York University, New York Post Graduate Medical School and Bellevue Hospital Alumni

Northwestern University Alumni—Postgraduates, Residents and Staff

Dr. Walter R. Parker-Dr. Roy Bishop Canfield Alumni

Presbyterian Hospital of New York Ear, Nose and Throat Alumni

Receiving Hospital Ophthalmic Alumni (Detroit)

University of Illinois Otolaryngologic Alumni Association

University of Iowa Alumni

University of Minnesota Eye, Ear, Nose and Throat Alumni

University of Pennsylvania Alumni—Otolaryngology

University of Texas Medical Branch, Galveston, Texas & Baylor University College of Medicine, Houston, Texas—Otolaryngology

Washington University—Ophthalmology

Washington University Alumni—Otolaryngology

Wills Eye Hospital Society Alumni

Tickets for alumni functions on Wednesday evening will be on sale from Sunday morning at nine o'clock until two o'clock on Wednesday.

MEMORIAL DINNER

The Henner Memorial Dinner will be held on Thursday, October 13, at the Standard Club, 320 S. Plymouth Court, Chicago. Dinner will be at 7:00 p.m., with cocktails from 6:00 to 7:00.

Dr. Julius Lempert of New York and Dr. Francis L. Lederer of Chicago will be guest speakers.

The public is invited.

CANDIDATES--1960

The following candidates have applied for Fellowship in the American Academy of Ophthalmology and Otolaryngology and will be voted upon at the Annual Business Meeting, Thursday, October 13, 1960. Objections to the election of a candidate must be filed with the Executive Secretary-Treasurer previous to Saturday, October 8, 1960.

CANDIDATES CERTIFICATED BY BOARDS

Ophthalmology (OP)
Otolaryngology (ALR)

Alderman, George Carl, 1201 N. Calvert St., Baltimore 1, Md., ALR

Allain, James John, 902 E. Edmond St., St. Joseph, Mo., OP

Allen, George Whitaker, 303 E. Chicago Ave., Chicago 11, Ill., ALR

Anhalt, Edward F., 713 Columbia St., New Westminster, B. C., Canada, OP

Anison, George C., 33 Medical Arts Sq. N.E., Albuquerque, N. M., ALR

Appleton, Budd, U. S. Army Hospital, Fort Hood, Texas, OP

Archer, Dewey Dale, 1901 Oak Park Blvd., Lake Charles, La., OP

Aronberg, Charles, 6360 Wilshire Blvd., Los Angeles 48, Calif., OP

Audette, Paul John, 266 Bridge St., Springfield 2, Mass., OP

Bade, Craig Perkins, 2006 N. Navarro, Victoria, Texas, OP

Bailey, Arby Lee, 98 Clinton St., Greenville, Pa., OP

Bailey, Norman Loudon, 215 Hammond Bldg., Moose Jaw, Sask., Canada, OP

Bailey, Paul Francis, Jr., 1216 S.W. Yamhill St., Portland 5, Ore., OP

Barber, Hugh Otter, 170 St. George St., Toronto 5, Ont., Canada, ALR

Barron, Stewart S., 4020 Junius St., Dallas 10, Texas, OP

Becker, Barton Robert, 1521 S. King St., Honolulu 14, Hawaii, ALR

Becker, Stanley Clifford, 3720 Washington Blvd., St. Louis 8, Mo., OP

Berard, William Peter, 1509 Medical Dental Bldg., Seattle 1, Wash., ALR

Berger, Ben, 1541 W. Broadway, Vancouver 9, B. C., Canada, OP

Bivins, Thomas E., 1209 Park Ave., San Jose 26, Calif., OP

Blum, Fred G., Jr., 110 E. Main St., Madison 3, Wis., OP

Boomer, Robert Bruce, 850 Middlefield Rd., Palo Alto, Calif., OP

Borkenhagen, Robert Herbert, 13000 S. Maple Ave., Blue Island, Ill., ALR

Boyd, Herschell Hood, 26 103rd Ave. N.E., Bellevue, Wash., OP

Brandow, Edward Carpenter, Jr., 678 Madison Ave., Albany, N. Y., ALR

Brown, David V. L., 122 S. Michigan Ave., Chicago 3, Ill., OP

Brugge, Eugene Homer, 426 17th St., Oakland 12, Calif., OP

Buckwald, Albert I., 666 E. Santa Clara St., San Jose 12, Calif., ALR

Burhans, John Burton, 418 Medical Arts Bldg., Grand Rapids 2, Mich., OP

Burton, Robert Dale, 300 Homer Ave., Palo Alto, Calif., ALR

Bussey, Joe Leverett, 1201 W. Presidio, Fort Worth 3, Texas, OP

Byer, Norman Eugene, 21320 Hawthorne Blvd., Torrance, Calif., OP

Campbell, Francis Patrick, New York University Medical Center, New York, N. Y., OP

Carlson, Albert Eugene, 925 W. Ninth Ave., Albany, Ore., OP

Cassady, John Rue, 921 Lincolnway E., South Bend 18, Ind., OP

Chulavachana, Tavisak, Mahachai Rd., Bangkok, Thailand, OP

Cipcic, Joseph Anthony, Fourth Floor, Mercy Hospital, Pittsburgh 19, Pa., ALR

Clark, James Robertson, 34 N. Ash St., Ventura, Calif., ALR

Cole, Roger W., 1718 Hampton St., Columbia 1, S. C., OP

Cook, Thomas William, Jr., 2300 N. Rockton Ave., Rockford, Ill., OP

Cowan, Robert F., 279 Woolwich St., Guelph, Ont., Canada, OP

Cowger, Robert C., 202 N.W. Cedar, Beaverton, Ore., OP

Csovanyos, Laszlo, 198 East Ave., East Norwalk, Conn., ALR

Curtin, Victor Thomas, 1700 N.W. Tenth Ave., Miami 36, Fla., OP

Cutino, Rudolph Lewis, 155 Henry St., Brooklyn 1, N. Y., OP

Davis, Albert James, 2100 Ninth St. South, St. Petersburg 5, Fla., ALR

DeGolia, Pershing, 858 Fourth St., Santa Rosa, Calif., OP

de Juan, Eugene, 1006 Merchants Nat'l Bank Bldg., Mobile, Ala., OP

Dellaporta, Angelos, 2000 Van Ness Ave., San Francisco 9, Calif., OP

Demming, James Henry, 100 Broadway, Daytona Beach, Fla., OP

Depp, Donald Samuel, 305 Royal Hawaiian Ave., Honolulu 15, Hawaii, OP

Dinsdale, Howard Arthur, U. S. Army Hospital, Fort Jackson, S. C., OP

Draheim, Jerry W., 1838 Parkwood Ave., Toledo 2, Ohio, OP

Drews, Robert Carrel, U. S. Naval Hospital, Great Lakes, Ill., OP

Ehrenreich, Jack, 299 Alhambra Circle, Coral Gables 34, Fla., ALR

Eisenberg, Isadore Jack, 1520 N. Second St., Harrisburg, Pa., OP

Enrile-Tolentino, Leticia M., New Panaderos St., Sta. Ana, Manila, Philippines, OP

Espiritu, Romeo B., Philippines General Hospital, Manila, Philippines, OP

Evans, David J., 1629 Truxtun Ave., Bakersfield, Calif., OP

Fajardo, Romeo V., Wills Eye Hospital, Philadelphia 30, Pa., OP

Feria, Quirico A., Jr., 1200 Misericordia Sta. Cruz, Manila, Philippines, OP

Ferreira, Jose Antonio, H. Irigoyen 384, Cordoba, Argentina, OP

Ferrer, Jose Luis, 6900 W. Belmont Ave., Chicago 34, Ill., ALR

Finley, Ralph Monroe, Jr., 603 Weeks St., New Iberia, La., OP

Fitton, Richard H., Jr., 385 Weetamoe, Fall River, Mass., ALR

Ford, Donald Pierce, 5000 Montrose Blvd., Houston 6, Texas, OP

Ford, James C., 9730 Wilshire Blvd., Beverly Hills, Calif., OP

Fragola, Anthony F., 763 Montauk Highway, West Islip, N. Y., ALR

Frank, Gordon David, 3707 Gaston Ave., Dallas 10, Texas, OP

Freeman, Joseph, 245 S.E. First St., Miami 32, Fla., ALR

Furey, Joseph Aloysius, 309 Blue Bonnet Blvd., San Antonio, Texas, ALR

Galin, Miles A., 525 E. 68th St., New York 21, N. Y., OP

Garrett, Frank E., 510 S. Mathilda, Sunnyvale, Calif., OP

Garrett, Spencer Roberts, 1417 Lakeland Hills Blvd., Lakeland, Fla., OP

Garvey, Edward E., 450 Sutter St., San Francisco 8, Calif., ALR

Gates, Lawrence Keith, 52 N. First East St., Logan, Utah, OP

Gaukrodger, William Thomas, 180 Metcalfe Street, Ottawa 4, Canada, OP

Gazala, Joseph Richard, 1001 W. Franklin St., Richmond 20, Va., OP

Geadah, Fouad Adib, A.B.C. Bldg., Hamra St., Beirut, Lebanon, ALR

Goff, John Robert, Fargo Clinic, Fargo, N. D., OP

Goldsby, Henry Herman, Jr., 1600 Fairfield Ave., Shreveport, La., OP

Gonzalez-Jimenez, Enrique Raul, Padial 38, Caguas, Puerto Rico, OP

Goodner, Ernest Krum, 1500 Fifth Ave., San Francisco 22, Calif., OP

Greiner, Wallace R., 1112 Medical Dental Bldg., Seattle 1, Wash., ALR

Hadlund, Ralph Louis, U. S. Naval Hospital, San Diego 34, Calif., OP

Hall, Dean Morris, 1842 W. Lincoln Ave., Anaheim, Calif., ALR

Hamilton, Ralph Sherrill, 934 Exchange Bldg., Memphis 3, Tenn., OP

Hamrick, George Vincent, 404 Medical Arts Bldg., Charleston 1, W. Va., OP

Harper, Douglas Willis H., 2451A Bloor St., West, Toronto, Ont., Canada, OP

Harper, John Y., Jr., Lackland Air Force Hospital, San Antonio, Texas, OP

Hartstein, Jack, 7165 Delmar Blvd., St. Louis 30, Mo., OP

Hatch, Joseph Lloyd, 465 E. South Temple St., Salt Lake City 11, Utah, OP

Henry, Morriss Murphey, 36th Tactical Hospital, APO 132, New York, N. Y., OP

Herring, Emmett McDonald, Green Clinic, Box 1230, Hattiesburg, Miss., OP

Hildyard, Victor H., 4200 E. Ninth Ave., Denver 20, Colo., ALR

Hill, William R., 28 W. Scribner Ave., Du Bois, Pa., OP

Honan, Paul R., 1720 N. Lebanon St., Lebanon, Ind., OP

Houck, Virgil L., 90 Shenango St., Greenville, Pa., OP

Hudgel, Lawrence Eugene, 237 S. Conwell St., Casper, Wyo., ALR

Hurst, Gerald Winton, U. S. Naval Hospital, Great Lakes, Ill., ALR

James, Burton R., 2900 Fresno St., Fresno, Calif., OP

Jamison, William Arthur, 76 Branch Ave., Red Bank, N. J., ALR

Jayapathy, B., University Hospitals, Iowa City, Iowa, ALR

Jester, Royal F., Jr., 214 W. 25th St., Kearney, Neb., OP

Johnson, Tom Lamar, 406 American Nat'l Bank Bldg., Shawnee, Okla., OP

Jones, Earl Rivers, 161 W. Cheves St., Florence, S. C., ALR

Kalmon, Morris, 159 S. Main St., Akron 8, Ohio, ALR

Karp, Harrison Myron, 1593 Rugby Rd., Schenectady 8, N. Y., ALR

Kasner, David, Apt. 8, 2794 S.W. 30th Ave., Miami 33, Fla., OP

Keays, Claude Fraser, 324 Spring Garden Rd., Halifax, N. S., Canada, OP

Keefe, Robert Joseph, 2345 Ocean Ave., San Francisco 27, Calif., OP

Kern, Arthur Stephen, U. S. Naval Hospital, Corpus Christi, Texas, OP

Khoury, Assad S., Washington Clinic, Wisconsin at Western Ave. N.W., Washington 15, D. C., ALR

Kinosian, Henry John, 323 Washington St., Ogdensburg, N. Y., OP

Kodros, Andreas George, 30 N. Michigan Ave., Chicago 2, Ill., ALR

Kohler, Henry Julius, 5118 N. Broad St., Philadelphia 41, Pa., OP

Kohtiao, Augusto Genato, 344 W. 72nd St., New York 23, N. Y., OP

Krcmery, Emil, 6005 W. Cermak Rd., Cicero 50, Ill., ALR

Kupfer, Carl, 243 Charles St., Boston 14, Mass., OP

Land, James Franklin, 110 E. Main St., Madison 3, Wis., ALR

Lang, Robert Muir, Russell Eve Bldg., Hamilton, Bermuda, ALR

LaPointe, Gaston A., 627 E. Jacques Cartier, Chicoutimi, Que., Canada, OP

Lawrence, Van Lloyd, 907 Hermann Professional Bldg., Houston 35, Texas, ALR

Levieff, Leo, 2875 Main St., Stratford, Conn., OP

Liddicoat, Douglas Arthur, 408 E. Lake, Watsonville, Calif., OP

Little, James Graham, Montreal General Hospital, 1650 Cedar Ave., Montreal 25, Que., Canada, OP

Livant, Saul, U. S. Veterans Administration, 35 Ryerson St., New York, N. Y., OP

Lowe, Percy Elliott, 1616 Memorial Professional Bldg., Houston 2, Texas, OP

Lubeck, Marvin Jay, Box 20, William Beaumont Army Hospital, El Paso, Texas, OP

Lundblad, Robert M., 307 S. 12th Ave., Yakima, Wash., OP

Lyon, Ernst F., 133 E. 58th St., New York 22, N. Y., OP

Lyons, George Dewey, 336 N. Jefferson Davis Blvd., New Orleans 19, La., ALR

MacLean, Robert George, 246 W. Main St., Los Gatos, Calif., ALR

Magee, George Franklin, 15 E. First St., Reno, Nev., OP

Manieri, Frank Vincent, 6002 Eastern Ave., Baltimore 24, Md., ALR

Marandi, Kazem Ahmadi, 15 E. Biddle St., Baltimore 2, Md., ALR

Martin, Berkeley Hancock, Jr., 1805 Monument Ave., Richmond 20, Va., OP

Martin, Howard Franklin, U. S. Naval Hospital, Jacksonville 14, Fla., ALR

Mastman, Gary J., 3415th USAF Hospital, Lowry AFB, Denver, Colo., OP

May, William, Winnipeg Clinic, Winnipeg, Man., Canada, OP

Mazer, Irwin Henry, Room 102, Osborne Medical Bldg., Winnipeg 1, Man., Canada, ALR

McCabe, Brian Francis, University Hospital, Ann Arbor, Mich., ALR

McCarthy, James L., 6429 W. North Ave., Oak Park, Ill., OP

McClelland, Randall John, 1124 Broadway, Quincy, Ill., OP

McDonald, George, 17 E. 72nd St., New York 21, N. Y., OP

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Minor, Ralph Hugh, 1515 Pacific, Everett, Wash., OP

Monroe, Charles A., USPHS Hospital, Galveston, Texas, OP

Morledge, Charles Calvin, 310 Hart-Albin Bldg., Billings, Mont., OP

Nachman, Elias, 303 S. Main, Bluffton, Ind., OP

Nager, George T., The Johns Hopkins Hospital, Baltimore 5, Md., ALR

Nail, Benjamin Miles, Jr., 1002 Medical Towers, Houston 25, Texas, ALR

Nassif, Ramzy Iskandar, Dept. of Otolaryngology, University Hospitals, Iowa City, Iowa, ALR

Odeh-Nasrala, Nicolas, Edif. Capitalizadora 201, Tegucigalpa, D. C., Honduras, C. A., OP

Ostiguy, Marcel, 3707 Saint Hubert St., Montreal, Que., Canada, ALR

Palpal-latoc, Leonarda A., San Ildefonso, Ilocos Sur., Philippines, OP

Peabody, Sherman Milne, U. S. Naval Hospital 926, FPO, San Francisco, Calif., OP

Pearlstone, Arnold David, Navy 115, Box 36, FPO, New York, N. Y., OP

Peterson, Walter C., 1601 16th Ave., Seattle 22, Wash., OP

Pifer, William H., 19 N. Washington St., Winchester, Va., ALR

Plowman, Edward Morris, 711 Grand Ave., Hannibal, Mo., ALR

Portenar, Myron Anthony, 419 First St., Lakewood, N. J., OP

Pougiales, Mary Lomas Price, 915 Third Ave. S.E., Rochester, Minn., OP

Preston, Frank R., U. S. Navy Hospital, Camp Pendleton, Oceanside, Calif., OP

Proffitt, Jonas A., Medical Center, 704 W. Koenig, Grand Island, Neb., OP

Pryor, Chester Cornelius II, Womack Army Hospital, Fort Bragg, N. C., OP

Rayfield, Roy Clifton, 75th Station Hospital, Bad Cannstatt, APO 154, New York, N. Y., OP

Relihan, Donald A., 1014 Central Bldg., Wichita 2, Kan., OP

Rex, Eugene Braiden, 102 Lankenau Medical Bldg., Philadelphia 31, Pa., ALR

Riley, Bennet J., 2598 East St., Concord, Calif., OP

Robinson, Stanley T., 228 Meridian Rd., San Jose, Calif., OP

Rosenbaum, Joseph George, 2460 Fairmount Blvd., Cleveland Heights 6, Ohio, OP

Rosenbluth, Joseph Carl, Second Field Hospital, APO 407, New York, N. Y., OP

Rowell, Peter Paul, 201 Livesley Bldg., Salem, Ore., OP

Sabri, Joseph Aziz, 1780 Massachusetts Ave, Washington 6, D. C., ALR

Sanders, Harmon U., 10901 Winner Rd., Independence, Mo., OP

Santos, Milagros T., Malabon, Rizal, Philippines, ALR

Sarin, Lov K., Wills Eye Hospital, Philadelphia 30, Pa., OP

Sawyer, Thomas Rarick, 1211 21st Ave., Nashville, Tenn., OP

Schulz, Kurt Johannes, 4655 Broadway, Gary, Ind., OP

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Scott, John Kimball, 110 E. Main St., Madison 3, Wis., ALR

Seeger, Joseph Gregory, USAF Hospital Chanute, Chanute AFB, Rantoul, Ill., OP

Shane, Arthur Gerald, 52 Quinpool Rd., Halifax, N. S., Canada, ALR

Shapiro, Lawrence Allen, U. S. Naval Base Hospital, Charleston, S. C., ALR

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Shoff, Mahlon James, Alaska Native Service Hospital, Anchorage, Alaska, OP

Simel, Paul J., EENT Clinic, U. S. Army Hospital, Fort Sill, Okla., OP

Simpson, Derek Grey, 925 W. Georgia St., Vancouver 1, B. C., Canada, OP

Singer, Robert Louis, 2000 Peralta Blvd., Fremont, Calif., OP

Singleton, George Terrell, 2207 Avondale, Wichita Falls, Texas, ALR

Siribodhi, Chira, 4654 Hazel Ave., Philadelphia 43, Pa., ALR

Smart, Don M., 9323 Garland Rd., Dallas 18, Texas, OP

Smersh, Thomas C., 1120 Cherry, Seattle 4, Wash., ALR

Smith, Cody Lane, 125 Prescott, El Cajon, Calif., OP

Smith, William B., 239 Pipestone St., Benton Harbor, Mich., ALR

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Soll, David Benjamin, USPHS Hospital, Seattle 14, Wash., OP

Sorenson, Roger William, Box 170, Valley Forge General Hospital, Phoenixville, Pa., OP

Stealey, Robert Lynn, 1938 Del Monte Dr., Findlay, Ohio, OP

Stern, Charles, Medical Arts Bldg., Natchez, Miss., OP

Sternstein, Herman Jerome, 54 Winter St., Norwood, Mass., ALR

Stevenson, Edward Ward, 1121 Somerville Rd. S.E., Decatur, Ala., ALR

Stewart, C. Thomas, Suite 1559 Medical Dental Bldg., Seattle 1, Wash., OP

Stothers, Hilton Hedley, 140 E. 54th St., New York 22, N. Y., ALR

Stroud, Malcolm Herbert, 640 S. Kingshighway, St. Louis 10, Mo., ALR

Sweebe, Edward Chiles, 243 Charles St., Boston 14, Mass., OP

Sykes, John H. J., 414 Navarro St., San Antonio 5, Texas, OP

Tashma, Albert, 1200 N. Vermont Ave., Los Angeles 29, Calif., OP

Thompson, Hardy Evans, Jr., 706 W. 19th St., Austin 1, Texas, OP

Tipshus, Alfons Fabian, 645 W. Harding Way, Stockton 4, Calif., OP

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Ural, Ibrahim Tali, Asagi Haman Mahallesi No. 7, Divrigi-Sivas, Turkey, ALR

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Utens, Max, 10092 Chapman Ave., Garden Grove, Calif., OP

van Fossen, Albert Wayne, 700 Bryden Rd., Columbus 15, Ohio, OP

Vincent, Bayard R., 1202 Delaware Ave., Wilmington, Del., OP

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Weatherall, Richard Hooper, USAF Hospital Wiesbaden, Box 330, APO 633, New York, N. Y., OP

Weimer, John Russell, Suite 202, 800 Pershing Dr., Silver Spring, Md., OP

White, Melvin Jeffrey, USAF Hospital, MacDill AFB, Tampa, Fla., OP

Wierzbinski, Francis Albert, 1241 N. Broadway, Billings, Mont., ALR

Wiesinger, Herbert, 2015 Monument Ave., Richmond 20, Va., OP

Wilder, William Wiggins, Suite 6, 4623 Poplar Ave., Memphis 17, Tenn., OP

Williams, Albert Harold, 109 Prichard St., Fitchburg, Mass., ALR

Williams, Barney Kenneth, Jr., Peterson Memorial Hospital, Kerrville, Texas, OP

Williams, Neil S., 1702 13th St. South, Fargo, N. D., OP

Wladecki, William B., 100 E. Broad St., Elyria, Ohio, ALR

Wootton, James Chipman, USAF Hospital, Maxwell AFB, Ala., OP

Wuerschmidt, Marcus Aurelius, 43 E. Bridge St., Oswego, N. Y., OP

Zauderer, Seymour J., 44 Gramercy Park North, New York 10, N. Y., ALR

The following candidates have qualified for Fellowship in the Academy except in some single detail, such as certification or the signatures of sponsors. The deficiencies are expected to be made up before the Annual Business Meeting.

Appalanarasayya, Kasibhatla, c/o K. V. Rao, Kottapata, Vizianagram, Andhra Pradesh, India, OP

Barraquer, Joaquin, Muntaner 314, Barcelona, Cataluna, Spain, OP

Binion, Warren Whiteley, 1201 W. Presidio St., Fort Worth 3, Texas, OP

Casas Hernandez, Rafael Augusto, Policlinica D'Empaire, Maracaibo, Zulia, Venezuela, OP

Cohen, Abraham Albert, 130 E. 67th St., New York 21, N. Y., ALR

Consul, Bishan Nath, Sawai Man Sing Medical College, Jaipur, Rajasthan, India, OP

Cooper, Rustom A. F., Naosari Bldg., D. Naoroji Rd., Bombay 1, India, ALR

Dhanda, Rajendra Pal, 58 New Palasia, Indore, M. P., India, OP

Grom, Edward, Instituto Medico del Este, Ave. Casanova, Caracas, Venezuela, OP

Malbran, Enrique, Parera 94, Buenos Aires, R. Argentina

O'Mahony, John B., 327 N. Christina St., Sarnia, Ont., Canada, ALR

Perez, Alfredo Celis, Edificio Celis Perez, Valencia, Venezuela, ALR

Tonndorf, Juergen, Medical Research Center, Iowa City, Iowa, ALR

Valcarce, Joaquin, Nunez de Balboa 74, Madrid, Spain, OP

FOR ASSOCIATE MEMBERSHIP

Raju, Datla Virabhadra, Pyna, Kakinada Taluk, Andhra State, India. M.D., Resident in Ophthalmology

Suie, Ted, Dept. of Ophthalmology, Ohio State University, Columbus, Ohio. Ph.D. Ophthalmic Microbiology and Immunology

PROGRAM IN BRIEF

Palmer House — Chicago

1960 Meeting

SATURDAY, OCTOBER 8

9:00 a.m. — Council Meeting — Private Dining Room 4

SUNDAY, OCTOBER 9

9:00 a.m.—Registration—Foyer to Grand Ballroom

9:00 a.m.—Ladies' Registration—Room 832

9:00 a.m.—Official Opening of Exhibits: Scientific—Private Dining Rooms 15, 16, 17; Club Foyer
Technical—Exhibition Hall and Red Lacquer Room, and Seventh Floor

10:00 a.m.—Teachers' Sections: Ophthalmology—Grand Ballroom

10:00 a.m.—Teachers' Sections: Otolaryngology—Crystal Room

12:30 p.m.—Home Study Faculty Luncheon—Private Dining Room 9

4:30 p.m.—President's Reception—Grand Ballroom

8:00 p.m.—Special Scientific Program: Otosclerosis Study Group of the Committee on Conservation of Hearing—Grand Ballroom

8:00 p.m.—Special Scientific Program: American Orthoptic Council and American Association of Orthoptic Technicians—Waldorf Room, Conrad Hilton Hotel

MONDAY, OCTOBER 10

9:00 a.m.—Orthoptic Technicians Instruction Courses—Seventh Floor

9:30 a.m.—Joint Scientific Session—Grand Ballroom

10:15 a.m.—Orthoptic Technicians Instruction Courses—Seventh Floor

11:30 a.m.—Orthoptic Technicians Instruction Courses—Seventh Floor

2:00 p.m.—Section on Otolaryngology: Scientific Papers and Motion Pictures —Grand Ballroom

2:00 p.m.—EYE Instruction Courses—Seventh Floor and Crystal Room

3:15 p.m.—EYE Instruction Courses—Seventh Floor and Crystal Room

4:30 p.m.—EYE Instruction Courses—Seventh Floor and Crystal Room

7:00 p.m.—Annual Banquet—Grand Ballroom

TUESDAY, OCTOBER 11

9:00 a.m.—Section on Ophthalmology: Scientific Papers and Motion Pictures —Grand Ballroom

9:00 a.m.—ENT Instruction Courses—Seventh Floor and Crystal Room

9:00 a.m.—Orthoptic Technicians Instruction Courses—Seventh Floor

10:15 a.m.—ENT Instruction Courses—Seventh Floor and Crystal Room

10:15 a.m.—Orthoptic Technicians Instruction Courses—Seventh Floor

11:30 a.m.—ENT Instruction Courses—Seventh Floor

11:30 a.m.—Orthoptic Technicians Instruction Courses—Seventh Floor

2:00 p.m.—Section on Otolaryngology: Scientific Papers and Motion Pictures —Grand Ballroom

2:00 p.m.—EYE Instruction Courses—Seventh Floor and Crystal Room

3:15 p.m.—EYE Instruction Courses—Seventh Floor and Crystal Room

4:30 p.m.—EYE Instruction Courses—Seventh Floor and Crystal Room

5:30 p.m.—Dinner Meeting of International Association of Secretaries of Ophthalmological and Otolaryngological Societies—Private Room 18

8:00 p.m.—Special Scientific Program: Committee on Plastic Surgery, Ophthalmology—Grand Ballroom

8:00 p.m.—Special Scientific Program: Committee on Plastic Surgery, Otolaryngology—Crystal Room

WEDNESDAY, OCTOBER 12

9:00 a.m.—Section on Ophthalmology: Scientific Papers and Motion Pictures —Grand Ballroom

9:00 a.m.—ENT Instruction Courses—Seventh Floor and Crystal Room

10:15 a.m.—ENT Instruction Courses—Seventh Floor and Crystal Room

11:30 a.m.—ENT Instruction Courses—Seventh Floor

12:45 p.m.—Council Meeting Luncheon—Private Dining Room 5

2:00 p.m.—Section on Otolaryngology: Scientific Papers and Motion Pictures—Grand Ballroom

2:00 p.m.—EYE Instruction Courses—Seventh Floor and Crystal Room

3:15 p.m.—EYE Instruction Courses—Seventh Floor and Crystal Room

4:30 p.m.—EYE Instruction Courses—Seventh Floor and Crystal Room

6:30 p.m.—Alumni Functions, unless otherwise noted

THURSDAY, OCTOBER 13

9:00 a.m.—Section on Ophthalmology: Scientific Papers and Motion Pictures —Grand Ballroom

9:00 a.m.—ENT Instruction Courses—Seventh Floor

10:15 a.m.—ENT Instruction Courses—Seventh Floor

11:30 a.m.—ENT Instruction Courses—Seventh Floor and Crystal Room

12:30 p.m.—Faculty Buffet Luncheon—Private Dining Room 14

2:00 p.m.—Section on Otolaryngology: Scientific Papers and Motion Pictures —Grand Ballroom

2:00 p.m.—EYE Instruction Courses—Seventh Floor

3:15 p.m.—EYE Instruction Courses—Seventh Floor

4:30 p.m.—EYE Instruction Courses—Seventh Floor

5:30 p.m.—Business Meeting—Private Dining Room 18

FRIDAY, OCTOBER 14

9:00 a.m.—Section on Ophthalmology: Scientific Papers and Motion Pictures —Grand Ballroom

RESERVE YOUR TABLE FOR THE ANNUAL BANQUET

All tables reserved. Tickets are on sale in the registration foyer.

EXCELLENT CUISINE ——— DELIGHTFUL ENTERTAINMENT

DANCE TEAM — NICKI AND NOEL

A NIGHT WITH SIGMUND ROMBERG

Get your tickets and choose your table. Make up your party and meet early for cocktail service in the banquet hall. Doors open at 6:30 p.m., banquet at 7:30 p.m. Dancing to follow, ten to twelve — NORM KRONE and his orchestra.

Tickets — $12.50

SCIENTIFIC SECTIONS

JOINT SESSION

Monday, October 10, 1960

9:30 a.m.

GRAND BALLROOM

Presiding Officer:

DEAN M. LIERLE, M.D.
President

Secretary:

CLAIR M. KOS, M.D.

Call to Order.....The President

Announcements

Introduction of the Guest of Honor
Gordon D. Hoople, M.D.
Syracuse, New York

Address by the President
Our Specialty Boards

Address by the Guest of Honor
The Pursuit of Excellence

Presentation of Honor Awards

RECIPIENTS OF HONOR AWARDS FOR 1960

Joseph P. Atkins
Philadelphia

Raymond Carhart
Evanston, Illinois

Paul L. Cusick
Detroit

Lester T. Jones
Portland, Oregon

Frank Lathrop
Boston

Mortimer Mann
Indianapolis

Don Marshall
Kalamazoo, Michigan

Carroll R. Mullen
Philadelphia

Kenneth M. Ogle
Rochester, Minnesota

Peter N. Pastore
Richmond, Virginia

Walter Z. Rundles, Sr.
Flint, Michigan

Robert E. Ryan
St. Louis

Albert P. Seltzer
Philadelphia

Emanuel M. Skolnik
Skokie, Illinois

Richard C. Troutman
New York

Linden J. Wallner
Chicago

Lorenz E. Zimmerman
Washington, D. C.

VIRUS TO MR. VIRUS
(A Motion Picture)

The Eye, Ear, Nose and Throat Research Division of the Doho Chemical Corporation with the cooperation of the Instituto Nazionale Luce and the Instituto Superiore Disanta of the Government of Italy have the honor to present for the first time and visible to the naked eye the growth of virus living cultures and the mechanics of propagation of the virus through the destruction of the individual cell.

SYMPOSIUM: EPIDEMIOLOGY AND CONTROL OF HOSPITAL INFECTIONS

Infections in the Operative Pavilion

Ralph Adams, M.D.
Wolfeboro, New Hampshire
BY INVITATION

The most important factor in control of operating room and hospital infections is cleanliness. Host-receptivity and resistance, and organism virulence are of great importance also. Walls, furniture, equipment, and floors of critical areas must be cleaned with an effective disinfectant-detergent, frequently repeated. Maintenance requires reduction of new sources of bacteria, through dust, fomites, and droplets, to a minimum. Air protection is afforded by an efficient air conditioning system, delivering air under positive pressure, with an exchange every four to twelve minutes, as related to occupancy. The principal cause of air contamination, human exhalation, is 90 to 95 per cent controllable by use of properly fitted filter masks. It is essential for personnel entering a critical area to put on fresh clean clothing over all skin surfaces. The skin of patients should be similarly covered, except for area to be prepared for surgery. Preoperative preparation of the patient's skin should be made by a 10-minute detergent-antiseptic solution scrub. The skin is then covered with a transparent plastic adherent film, similar in effect to the rubber gloves worn by surgeon and personnel.

Management of Infections Elsewhere in the Hospital

Leighton E. Cluff, M.D.
Baltimore
BY INVITATION

A surveillance of staphylococcal infection acquired by patients hospitalized at The Johns Hopkins Hospital during 1959 was made, and the data obtained emphasize that knowledge of the overall incidence of infection does not reflect the nature of the problem. It has been found, for example, that groups of patients most susceptible to infection include those with cancer, burns, dermatitis, diabetes mellitus, lupus erythematosus and cirrhosis of the liver, and newborn infants and post partum mothers. Similarly, it appears that certain types of surgical procedures are associated with a higher incidence of postoperative wound infection. The impact of these findings on methods of control or prevention of infection in the hospital will be discussed. In addition, a succinct review of the modes of transmission of staphylococci of possible importance in infection will be considered, and some of our studies on transmission by the airborne route and by direct contact will be presented.

Bacteriology in Relation to Hospital Infections

Henry F. Allen, M.D.
Boston

Hospital Infections from the Viewpoint of the Hospital Administrator

Dean A. Clark, M.D.
Boston
BY INVITATION

SECTION ON OTOLARYNGOLOGY

Monday, October 10, 1960

2:00 p.m.

GRAND BALLROOM

Presiding Officer:

DEAN M. LIERLE, M.D.
President

Secretary:
CLAIR M. KOS, M.D.

Presentation of Awards by Committee on Research in Otolaryngology

RECIPIENTS OF AWARDS

George W. Allen, M.D.
F. R. Kirchner, M.D.
A. Panagopoulos, M.D.
Frank N. Ritter, M.D.
George T. Singleton, M.D.

2:05 p.m.

MAN RETURNS TO THE SEA
(A Motion Picture)

G. Dekle Taylor, M.D.
Jacksonville, Florida

Millions of years ago man evolved from the sea. He is now a terrestrial being, and his anatomy and physiology are not suited to an aquatic environment. For thousands of years he has contemplated returning to the sea. Centuries of imagination, endeavor, and patient experimentation were necessary, however, before his ingenuity enabled him to adapt himself again to the underwater world. He has few of the structural adaptations for an aquatic or subaquatic environment that are seen in the porpoise, pilot whale, hippopotamus, and other aquatic animals. This film shows man's adaptation to the water by proper methods of breathing and bathing. The evolution of the various swimming strokes, face mask, snorkel, and self-contained underwater breathing apparatus is shown. The effect of pressure on the ear and sinuses is demonstrated. If man will understand and accept the limitations Nature has placed on him in an aquatic environment, he is now able to return to the sea.

2:35 p.m.

SURGICAL ALTERATIONS OF THE LARYNGEAL LYMPHATICS

John J. Welsh, M.D.
Louis W. Welsh, M.D.
Frank M. Behlke, M.D.
Iowa City
BY INVITATION

Discussers:

F. Johnson Putney, M.D.
Philadelphia

Walter H. Maloney, M.D.
Cleveland

The occurrence of cervical metastases from carcinoma of the larynx has stimulated further investigation into the normal lymphatic drainage of the larynx. The occasional contralateral metastasis appears in sufficient numbers to study the mechanism by which this event takes place.

A normal group of specimens was compared with a second series which had previously undergone radical cervical lymphatic dissection. Using radioactive tracer technique, the endolaryngeal areas have been investigated and correlation drawn between the injection site and the cervical lymph nodes involved with drainage.

External scintigraphy provides the approximate location of the nodal depot sites of drainage. More precise measurement of the tissue activity yields quantitative information concerning the lymphatic function.

Minute amounts of colloidal radio gold have been utilized in this study. The injections were made perorally into the cordal and supraglottic areas, and via tracheotomy into the infraglottic larynx. This approach affords expanse of the larynx and exact localization of instillation.

The finding that the drainage is radically altered from the normal will be illustrated by appropriate scintigraphs and histograms.

3:10 p.m.

EXPERIMENTAL OTOTOXICITY WITH STREPTOMYCIN AND DIHYDROSTREPTOMYCIN

T. Manford McGee, M.D.
Detroit
BY INVITATION

Discusser:

George E. Shambaugh, Jr.
Chicago

This discussion deals with the clinical problems related to toxicity due to streptomycin and dihydrostreptomycin. The problems will be illustrated with case presentations, which demonstrate vestibu-

lar and cochlear dysfunction associated with streptomycin and dihydrostreptomycin. Of particular interest will be the presentation of cases which exhibited a delayed hearing loss following the use of dihydrostreptomycin.

To correlate with these clinical observations, a series of experimental animals (cats) which received toxic doses of of streptomycin and dihydrostreptomycin will be reviewed. The auditory and vestibular behavior of the animals will be followed, and the histopathology of the temporal bones and brain stem nuclei studied. This includes animals that suffered a delayed hearing loss after having been on toxic doses of dihydrostreptomycin.

3:35 p.m.

THE USE OF COMPUTERS IN STUDIES OF AUDITORY NEUROPHYSIOLOGY

Nelson Yuan-Sheng Kiang, M.D.
Boston

BY INVITATION

The various ways in which electronic computers have been used by members of the Communications Biophysics Group to study the electrical activity of the auditory nervous system will be described. The presentation will emphasize the nature of the scientific problems which impelled the use of specific computers. Particular emphasis will be placed on averaging techniques, variability studies, and problems in the analysis of single unit data. Examples will be given in each instance to show how the problem arose, how the characteristics of the problem determined the methods of data-processing and how these desiderata in turn dictated the requirements for the computer. It is hoped that from these "case histories" a certain general approach in dealing with such problems will become evident.

AUTHORS OF PAPERS

All slides or films must be in the hands of the projection operator before the opening of each session.

Please be prepared to submit papers to the recorder immediately following presentation.

4:05 p.m.

SEQUESTRATION CYSTS OF THE FLOOR OF THE MOUTH
(A Motion Picture)

Loring W. Pratt, M.D.
Waterville, Maine

This paper reviews the embryology of the floor of the mouth with particular reference to the inclusion of islands of ectodermal tissue in the floor of the mouth as the result of closure of the mandibular arches. The literature is reviewed, and two specific cases of unusually large epidermoid cysts in the floor of the mouth are presented. This is illustrated with a motion picture which shows the cyst preoperatively and demonstrates the patient's ability to move it around in his mouth. Surgical removal of the cyst is demonstrated, and the postoperative results of the operation are demonstrated.

4:30 p.m.

OBLITERATION OF THE CAVITY AND RECONSTRUCTION OF THE AUDITORY CANAL IN TEMPORAL BONE SURGERY

Frederick R. Guilford, M.D.
Houston, Texas

Elimination of the mastoid bowl, with its objectionable sequelae, may be routinely achieved in modified radical and radical-type mastoid and tympanoplastic surgery. The external auditory canal is reconstructed and the mastoid cavity obliterated. Combined endaural and postauricular incisions are used. When it is necessary to exenterate the mastoid cells and remove the posterior bony wall of the external auditory canal, the technique is employed. The method may be used in conjunction with single or multiple stage tympanoplasty as well as modified radical

or radical mastoidectomy. Cavities resulting from previously performed mastoidectomies, tympanoplasties and fenestrations have been obliterated.

Following the mastoidectomy, the tip of the mastoid process is removed when the bone is well pneumatized, or partially removed when the bone is of the diploic type, which may have a nonpneumatized mastoid tip. The postauricular skin is then undermined posteriorly and superiorly. A soft tissue flap of an appropriate size is formed from the subcutaneous tissue, fascia, sternomastoid muscle and lower portion of the temporal muscle by sharp dissection. The soft tissue pedicle is inferior, and the flap is formed principally by the sternomastoid muscle and its fibrous attachments. This flap is trimmed to obliterate the mastoid bowl from its lower extremity up to the tegmen just posterior to the lateral semicircular canal. Occasionally a flap of temporal muscle with its pedicle posteriorly is used to aid in obliterating the sinodural angle. Appropriate packing is added to the external auditory canal area, the incisions closed and a mastoid dressing applied.

A skin flap from the external auditory canal partially covers the posterior wall of the external auditory canal, which is now formed by the muscle flap. The tegmen medially and soft tissue laterally form the roof of the canal. A delayed split-thickness skin graft is applied to the unhealed portion of the canal as an office procedure eight to ten days postoperatively.

The technique will be discussed, and a preliminary report of its use in 40 cases will be given.

SECTION ON OPHTHALMOLOGY

Tuesday, October 11, 1960

9:00 a.m.

GRAND BALLROOM

Presiding Officer:

HAROLD G. SCHEIE, M.D.
Vice-President

Secretary:
KENNETH L. ROPER, M.D.

CHONDROSARCOMA OF THE ORBIT

(A Clinicopathologic Study of Two Cases)

Monte G. Holland, M.D.
Herbert Ichinose, M.D.
BY INVITATION
and
James H. Allen, M.D.
New Orleans

Chondrosarcomas of the bones of the orbit are "exceedingly rare," according to Duke-Elder.

The authors present two cases of chondrosarcoma arising from the orbital bones. The presenting complaint in each case was exophthalmos. The clinical course, with illustrations of the appearance of the patients and the histologic character of the tumors, is presented. The patients showed a strikingly similar clinical picture. The treatment and subsequent course of the patients is discussed.

This very rare disease should be of interest to both ophthalmologists and otolaryngologists.

9:10 a.m.

ON APPLANATION TONOMETRY

Dr. Theo. A. F. Schmidt
Bern, Switzerland
BY INVITATION

9:30 a.m.

THE REGISTRY OF OPHTHALMIC PATHOLOGY: PAST, PRESENT and FUTURE

(XVII Jackson Memorial Lecture)

Lorenz E. Zimmerman, M.D.
Washington, D. C.

10:15 a.m.

EXCISION OF IRIS MELANOMA

(A Motion Picture)

Algernon B. Reese, M.D.
New York

This is a composite film of the technique employed in three excisions of iris melanomas. It shows types of incision and manner of performing the basal iridectomy in order to prevent seeding.

10:35 a.m.

SURGICAL MANAGEMENT OF SECONDARY EXOTROPIA

Edmond L. Cooper, M.D.
Detroit

Discusser:

Rudolf Aebli, M.D.
New York

Because the results of ocular muscle surgery are hard to predict, operations for esotropia sometimes result in overcorrection. There are relatively few references to overcorrection in the literature, and most authors who mention it discuss only the etiologic factors involved. Those authors who refer to management emphasize that reoperation should be on the same muscle, that the operation is usually difficult, and that it must be more extensive than the first operation.

A study of cases in which overcorrection of the deviation resulted from an operation for esotropia shows that the overcorrection may have certain characteristics which point the way to proper management. When an operation for esotropia results in secondary exotropia, the exotropia often measures greater at one distance of gaze than another.

Whenever overcorrection results from ocular muscle surgery, the case should be re-evaluated on the basis of diagnostic findings following the overcorrection. The surgeon should plan his approach to the new problem just as though no previous surgical intervention had occurred. It frequently is advisable to revise an overcorrection by an attack on previously unoperated muscles; the idea of always "undoing what was done" should be rejected.

11:05 a.m.

PLEOPTICS

Arthur Linksz, M.D.
New York

Discusser:

Hermann M. Burian, M.D.
Iowa City

New instruments, especially new ophthalmoscopes, have been made available for the diagnosis and treatment of eccentric fixation in amblyopia. The optics of these ophthalmoscopes are not generally understood. Once the principles of their construction are clarified, it becomes possible to adjust some of the other types of ophthalmoscopes for these special purposes.

11:30 a.m.

THE MUSHROOM CORNEAL GRAFT (A Motion Picture)

R. Winston Roberts, M.D.
Winston-Salem, North Carolina

The history of and indications for the mushroom or step-type corneal transplant operation will be briefly presented. A system of instruments and a technique for their use have been devised to make this technically formidable operation relatively safe, accurately reproducible, and easy to perform. These instruments and the technique will be described by means of a film which will include the actual performance of the mushroom corneal graft operation on a human patient.

11:50 a.m.

A MAJOR CAUSE OF HYPOTONY

Paul A. Chandler, M.D.
Boston
A. Edward Maumenee, M.D.
Baltimore

Discusser:

John H. Dunnington, M.D.
New York

The specific cause of hypotony following operative procedures, trauma, and certain intraocular inflammation is not always readily apparent. In some instances a fistula from the anterior chamber to the exterior of the eye causes an abnormal lowering of the intraocular pressure. There are many hypertonic eyes, however, without such a fistula. Evidence will be

cited that suggests that in at least some of these cases the hypotony is due to hyposecretion of the aqueous caused by a detachment of the choroid and ciliary body.

The evidence that a detachment of the pars plana of the choroid and ciliary body produced a hyposecretion of aqueous is as follows:

1. Localized small areas of detachment are found in this region in practically every eye examined histologically where the tension has been exceedingly low prior to enucleation. This is true even when a cyclitic membrane is not present to pull the ciliary body inward.

2. The anterior chamber becomes shallow in eyes that are hypotonic following cyclodialysis.

3. It can be demonstrated that the circulation of the aqueous is decreased in an eye with hypotony by the intravenous injection of fluorescein.

4. Fluorescein injected into the eye with hypotony following cyclodialysis has been recovered from an incision through the sclera 2 mm. from the limbus and 180 arc degrees away from the cyclodialysis cleft.

5. Fluid has been found over the ciliary body in hypotonic eyes when a cyclodialysis cleft is closed or when there has been excessive filtration from an external fistulizing operation for glaucoma. In these instances fluorescein injected into the anterior chamber did not diffuse into the superchoroidal fluid.

6. Reattachment of the ciliary body to the sclera has resulted in an increase in intraocular pressure.

SECTION ON OTOLARYNGOLOGY

Tuesday, October 11, 1960

2:00 p.m.

GRAND BALLROOM

Presiding Officer:
DAVID R. HIGBEE, M.D.
Vice-President

Secretary:
CLAIR M. KOS, M.D.

EXPERIMENTAL STUDIES ON SOME LITTLE UNDERSTOOD ASPECTS OF THE PHYSIOLOGY OF THE RESPIRATORY TRACT AND THEIR CLINICAL IMPORTANCE

(XVI Wherry Memorial Lecture)

Anderson C. Hilding, M.D.
Duluth, Minnesota

2:30 p.m.

PSYCHOLOGICAL CONSIDERATIONS IN THE MANAGEMENT OF CANCER OF THE EAR, NOSE AND THROAT

Daniel Miller, M.D.
Boston

This paper concerns itself with the decisions made in regard to irradiation or surgery, the amount of surgical intervention or the amount of irradiation deemed necessary for a specific individual with a specific type of cancer and a specific location. It includes a discussion of the effect of this decision, not only on the patient, but on his family and his fellow citizens. It concerns itself with a decision on how radical one should dare to be in a given case as well as with the decision to do very little, and to choose palliation rather than an attempted cure. It suggests the areas of soul-searching we all must do as otolaryngologists who treat cancer.

3:00 p.m.

TRAUMATIC INTRACRANIAL VASCULAR LESIONS PRODUCING LATE MASSIVE NASAL HEMORRHAGE

Delmar F. Weaver, M.D.
and
Edward Gates, M.D.
BY INVITATION

Aage E. Nielsen, M.D.
BY INVITATION
Detroit

A brief discussion will be given of nasal hemorrhage in general.

Most of the paper will be devoted to massive, and often fatal, nasal hemorrhage occurring from one week to six months following fracture of the cranium. The source of bleeding is the internal carotid artery. An aneurysmal sac has usually formed around a tear in the artery and this ruptures into the nose after eroding through bone or dissecting soft tissue.

Routes of entrance into the nose will be given.

The important factors regarding the cases that have been found in the literature will be tabulated, and two cases from the personal experience of the authors will be reported in some detail.

A discussion of diagnosis and treatment will follow.

3:30 p.m.

CINE-PANEL: PROBLEM CASES IN NEOPLASTIC AND TRAUMATIC DISEASE OF THE HEAD AND NECK

Moderator:

John F. Daly, M.D.
New York

Panelists:

John J. Conley, M.D.
New York

Joseph H. Ogura, M.D.
St. Louis

Edwin W. Cocke, M.D.
Memphis

William T. Moss, M.D.
Chicago
BY INVITATION

The presentation will be in the form of an interrupted motion picture in color giving the details of the history in each of six cases and illustrating the problem by close-ups of each patient. The moderator will guide the panelists in working out the best solution to the problems thus presented.

SECTION ON OPHTHALMOLOGY

Wednesday, October 12, 1960

9:00 a.m.

GRAND BALLROOOM

Presiding Officer:
JAMES H. ALLEN, M.D.
Vice-President

Secretary:
KENNETH L. ROPER, M.D.

WEGENER'S GRANULOMATOSIS (A Clinicopathologic Case Report)

William E. Borley, M.D.
and
William W. Miller, M.D.
BY INVITATION
San Francisco

Wegener's granulomatosis is a uniformly fatal syndrome characterized by necrotizing granulomatous lesions of the respiratory tract, generalized arteritis, and focal glomerulonephritis. Approximately 50 per cent have ocular involvement.

The case to be presented is that of a 37-year-old white male who suffered from unilateral chronic keratoconjunctivitis for two years. Prior to expiration, scleritis, uveitis, inflammatory retinal detachment, orbital tumor, and lung lesions developed. Treatment and autopsy findings will be discussed.

9:10 a.m.

TONOGRAPHY: A SYMPOSIUM

The Principle of Tonography: Terms and Definitions

Peter C. Kronfeld, M.D.
Chicago

Technique of Tonography: Typical Tonograms, Calculations and Interpretations

Elmer J. Ballintine, M.D.
Cleveland

Errors in Tonography

Robert A. Moses, M.D.
St. Louis

Experimental Tonography

W. Morton Grant, M.D.
Boston
BY INVITATION

Tonography in the Diagnosis of Chronic Simple Glaucoma

Bernard Becker, M.D.
St. Louis

Tonography in the Management of the Primary Glaucomas

R. Winston Roberts, M.D.
Winston-Salem, North Carolina

Summary

Peter C. Kronfeld, M.D.
Chicago

11:30 a.m.

UPPER LID RECONSTRUCTION WITH FREE SKIN GRAFT ON PEDICLE FLAP OF CONJUNCTIVA AND MUSCLE
(A Motion Picture)

Alston Callahan, M.D.
Birmingham, Alabama

In the reconstruction of a large defect of the upper lid, a free skin graft (for the anterior lamina) will survive well on a posterior lamina formed of a pedicle of conjunctiva orbicularis and some of the levator. The function and appearance of such a reconstructive lid is better than when the anterior lamina is formed by a pedicle flap transferred from the lateral canthal area.

In transferring tissue from the opposing lid in a Hughes procedure, the incision into the tarsus of the donor lid should not be made in the margin, but on the conjunctival surface at least 3 mm. from the posterior lip of the margin. This makes it easier to divide the lids properly after four months, when full contraction has occurred, and more important, this prevents irregular lid margins and makes preservation of the cilia in the donor lid more probable.

11:45 a.m.

PERCUTANEOUS RENAL BIOPSY AND OCULAR VASCULAR DISEASE
(Includes Short Motion Picture)

Frank W. Newell, M.D.
and
John D. Arnold, M.D.
BY INVITATION

Benjamin Spargo, M.D.
BY INVITATION

Bertha A. Klien, M.D.
Chicago

Discusser:
Lorenz E. Zimmerman, M.D.
Washington, D. C.

Percutaneous renal biopsy has been widely used in recent years in the diagnosis and study of a variety of kidney lesions. It provides data concerning the nature of the kidney abnormality prior to end-stage scarring and makes available material suitable for electron microscopy. The procedure is contraindicated in patients with a single kidney, blood dyscrasias, or malignant hypertension, and in those receiving anticoagulant therapy. Fever, renal infection, obesity and anasarca are not considered contraindications to the procedure.

The information provided by renal biopsy material has proved valuable in the diagnosis of subclinical diabetes with microaneurysms and chronic "glomerulonephritis" associated with the nodular lesion of Kimmelstiel and Wilson. In bilateral central venous closure, a similar renal venous abnormality was demonstrated. Renal changes of nephrosclerosis have been shown in youthful patients with retinal neovascularization. Renal biopsy provides a method of correlating vascular changes in the eye with renal changes prior to autopsy. A discussion of the technique of biopsy and findings will be presented.

SECTION ON OTOLARYNGOLOGY

Wednesday, October 12, 1960

2:00 p.m.

GRAND BALLROOM

Presiding Officer:
DEAN M. LIERLE, M.D.
President

Secretary:
CLAIR M. KOS, M.D.

PRESENTATION OF NEW INSTRUMENTS

2:30 p.m.

THE LARYNX AND VOICE: THE FUNCTIONS OF THE PATHOLOGIC LARYNX (A Motion Picture)

Hans von Leden, M.D.
and
Paul Moore, Ph.D.
BY INVITATION
Chicago

The third in a series of motion pictures on the larynx and voice, this film introduces the functional adjustment of the larynx to pathologic changes. It presumes a knowledge of the normal larynx, which was described in the two preceding films.

The broad area of laryngeal pathology and associated vocal changes is demonstrated through a series of patients with different laryngeal diseases. In each case the clinical appearance of the larynx and the related voice are presented synchronously for identification. These clinical manifestations are followed by brief examples of the underlying, basic physiology in ultraslow motion picture photographed at speeds up to 5,000 frames per second.

This cinematographic approach provides new and detailed information on vocal cord vibration and arytenoid motion in the pathologic larynx. Diagnostic or prognostic clues are offered in such different conditions as laryngeal edema, inflammations, and neoplasms. The pictorial evidence includes information of value in planning indicated therapy.

3:00 p.m.

THE PROTECTIVE VALUE OF THE STAPEDIUS REFLEX: AN EXPERIMENTAL STUDY

David A. Hilding, M.D.
Fort Knox, Kentucky
BY INVITATION

Discusser:

Scott N. Reger, Ph.D.
Iowa City

The purpose of this paper is to present further evidence on the protective function of the stapedius reflex, particularly as related to the phenomenon of protection afforded by a short pure tone preceding a loud impulse-type noise.

Cats were implanted with round window electrodes, permitting recording of cochlear potential response over a period of weeks.

A significant loss of sensitivity occurred in the ears exposed to the sound of gunfire unless they were given the benefit of a short pure tone immediately preceding each round.

If the stapedius was denervated or if its tendon was severed, there was no protection afforded by the tone. However, separating the tensor tympani from its attachment resulted in no significant loss of protection.

It appears that the "protective tone" acts by eliciting a reflex contraction of the stapedius muscle, and that this is effective in reducing acoustic trauma.

3:30 p.m.

EFFECT OF CIGARETTE SMOKE ON HUMAN RESPIRATORY CILIA (A Motion Picture)

John J. Ballenger, M.D.
Winnetka, Illinois

If it is accepted that heavy and prolonged cigarette smoking leads to an increased incidence of bronchogenic car-

cinoma, how is the toxic material deposited in the lung to initiate this process? This film demonstrates experimentally in vitro a mechanism whereby this may occur.

Minute explants of human respiratory ciliated cells are placed in a hanging drop preparation according to usual tissue culture techniques. Being very small, the beating of the cilia causes small aggregates of cells to rotate up to thirty times per minute. This rotation is used as the criterion of ciliary activity, and a motion picture record is made. If bathed in a balanced salt solution, beating and rotation are unimpaired and will continue several days. The salt solution is rendered toxic to the activity of the cilia simply by mechanically bubbling a small amount of cigarette smoke through it. The cilia quickly become inactive and rotation ceases if exposed to the smoke-salt solution.

Since the continuously beating cilia comprise the principal cleansing mechanism of the lung and nose, their paralysis or failure is of great importance to health. This film demonstrates that there is a toxic product in smoke from cigarettes which interrupts the ciliary cleansing mechanism. If the toxic smoke-balanced salt solution is promptly removed, the beating of the cilia tends to regain its former efficiency. Room air alone was not found to contain any material toxic to the cilia.

The conclusion suggested is that cigarette smoke interrupts the normal ciliary self-cleansing function of the lung and permits toxic material to accumulate on the bronchial surfaces. If the cleansing function is interrupted for long periods of time by heavy and prolonged smoking, a carcinoma may be initiated.

4:00 p.m.

SURGICAL TREATMENT OF MALIGNANT LESIONS OF THE NOSE AND SURGICAL RECONSTRUCTION

John Marquis Converse, M.D.
New York

Discusser:

William C. Huffman, M.D.
Iowa City

4:30 p.m.

A CLOSED METHOD OF SEMICIRCULAR CANAL FENESTRATION

J. H. Thomas Rambo, M.D.
New York

Discusser:

Merle Lawrence, Ph.D.
Ann Arbor, Michigan

Fenestration of the horizontal semicircular canal has the great advantage of bypassing the otosclerotic growth. This principle of avoiding the pathologic area in creating an avenue of sound transmission to the inner ear has resulted in a high percentage of lasting hearing improvement.

The disadvantage of the procedure has been the necessity for creating an "open radical cavity." The healing and aftercare of the fenestration cavity has been the main objection to the operation by both the patient and the surgeon.

The purpose of this paper is to describe a one-stage closed method of fenestration which eliminates the open postoperative cavity while retaining the great advantage of an extratympanic window that provides for a predictable and permanent hearing result.

The physiology of hearing in the closed fenestration and case results are briefly presented.

SECTION ON OPHTHALMOLOGY

Thursday, October 13, 1960

9:00 a.m.

GRAND BALLROOM

Presiding Officer:
HAROLD G. SCHEIE, M.D.
Vice-President

Secretary:
KENNETH L. ROPER, M.D.

NEVOXANTHOENDOTHELIOMA OF THE IRIS: DIAGNOSIS BY BIOPSY AND CURE WITH X-RAY
(A Clinicopathologic Case Report)

Gilbert W. Cleasby, M.D.
San Francisco

A six-month-old white girl was noted by her parents to have a yellow spot on the left iris. Shortly thereafter, two spontaneous anterior hemorrhages occurred.

Examination revealed an elevated intraocular pressure, hazy cornea, and a yellowish pink lesion over the inferior temporal quadrant of the left iris. Blood and flocculent tumor tissue were spread along the angle and on the back of the cornea.

An iris biopsy was performed, substantiating the clinical diagnosis of nevoxanthoendothelioma. Shortly thereafter, the cornea became more hazy, the entire iris became infiltrated with tumor tissue, and an iris prolapse occurred.

Roentgen therapy resulted in almost immediate regression of the tumor. Within one week the iris was nearly clear, the cornea was clear and the prolapse had disappeared.

Since the time of the treatment, the eye has remained apparently normal except for the small coloboma where tissue was removed for biopsy.

9:10 a.m.

SYMPOSIUM: PHOTOCOAGULATION

Dohrmann K. Pischel, M.D.
San Francisco
Graham Clark, M.D.
New York
Paul A. Cibis, M.D.
St. Louis

10:10 a.m.

BACTERIAL ALLERGY OF THE EYE

Frederick H. Theodore, M.D.
New York

Discusser:

Alson E. Braley, M.D.
Iowa City

Many of the important manifestations of ocular allergy appear to arise on the basis of hypersensitivity to products of bacteria and other micro-organisms. While such microbiallergic reactions play a definite role in disease of the conjunctiva and eyelids, they are even more important in the cornea and in the uvea. Many common forms of keratitis are of microbiallergic origin. The etiologic significance of bacterial allergy in uveal disease is generally accepted; however, allergic mechanisms in uveitis may be of a more complex character than heretofore suspected. The little we know of scleritis indicates that microbiallergic reactions are of major importance. Even in the retina and optic nerve, allergic reactions to microbial products have been reported. The mechanisms involved, the clinical manifestations of bacterial allergy in these structures, and their management, will be discussed.

10:45 a.m.

PATHOGENESIS OF NONAPPOSITIONAL GONIOSYNECHIAE: A NEW TECHNIQUE OF GONIO-CINEMATOGRAPHY
(A Motion Picture)

Adolph Posner, M.D.
New York

A technique has been developed for goniophotography with an 8 mm. motion picture camera coupled parfocally with a slit lamp. This method also lends itself to photography of the fundus and of the anterior segment. It facilitates comparative observations, so important in both diagnosis and treatment, and also offers a valuable teaching aid.

In the film are presented unusual cases to illustrate the manner in which peripheral anterior synechias form when there is no contact between the walls of the angle. These cases include essential atrophy of the iris, phacotoxic uveitis and posterior embryotoxon.

11:00 a.m.

TUMORS DEVELOPING AFTER RADIATION OF THE EYE

Arnold W. Forrest, M.D.
White Plains, New York

Lorenz E. Zimmerman, M.D.
Washington, D. C.

Discusser:

William F. Hughes, Jr., M.D.
Chicago

This report presents the details of a study of ten patients with bilateral retinoblastoma who were treated with radiation. In the exposed areas of the head a variety of mesenchymal and epidermal tumors developed at intervals after treatment ranging from four to twenty-seven years. An analysis of previously reported cases is annotated.

Several patients who have developed tumors about the eye following occupational radiation exposure or treatment of acne, eczema, or lymphosarcoma of the lacrimal sac are discussed.

11:30 a.m.

ALLERGIC REACTIVITY OF IRIS EPITHELIUM IN VITRO AS DEMONSTRATED BY INTERFERENCE CINEMICROGRAPHY (Including Motion Picture)

Charles D. Dukes, Ph.D.
BY INVITATION

Hilda H. Fox
BY INVITATION

Louis J. Girard, M.D.
Houston, Texas

For many years, it has been accepted that immediate-type hypersensitivity reactions are restricted to those areas involving the clinically demonstrable effects characterized by increased vascular permeability and smooth muscle contraction. However, many of the more subtle physiologic effects of immediate-type allergic reactions cannot be categorized as occurring secondarily to effects on endothelium or smooth muscle. With visual direct microscopy of tissue cultures, tissues obtained from allergically sensitized animals exhibited no demonstrable reaction when exposed to the specific allergenic antigen. When time-lapse cinemicrography is employed, it is possible to demonstrate that a specific allergic reaction takes place in sensitized tissues in culture. This reaction is subtle, but profound, and of such a deliberate, leisurely speed as to be unrecognizable until exposed by the compression of time made possible by the time-lapse equipment. When the duration of the reaction is compressed photographically from 100 to 200 times, a definite pattern of events occur in the allergic reaction in vitro. The pattern can be readily differentiated from those which occur following exposure of the same or of comparable normal tissues to irritative or to toxic stimuli. In addition, the pattern of events elicited during the allergic reaction were found to be mimicked by exposure to minute concentration of histamine. These results were obtained by the use or iris epithelium explants from guinea pigs sensitized to equine serum.

11:45 a.m.

NECROSIS OF IRIS, CILIARY BODY AND RETINA FOLLOWING RETINAL DETACHMENT OPERATIONS WITH CIRCLING POLYETHYLENE TUBE

Milton Boniuk, M.D.
BY INVITATION

Lorenz E. Zimmerman, M.D.
Washington, D. C.

Discusser:

Dohrmann K. Pischel, M.D.
San Francisco

Two eyes studied at the Armed Forces Institute of Pathology revealed extensive necrosis of the retina, ciliary body and iris following retinal detachment operations with circling polyethylene tube. Intraocular hemorrhage and mature cataracts developed after the operation, and in both cases atrophy and inflammation of the globe led to enucleation within three months.

The necrosis is considered to be the result of vascular damage. Factors responsible for this vascular insufficiency include detachment of the recti with severance of the anterior ciliary vessels and compression of branches of the vortex vein, with resultant edema of the choroid anterior to the tube. Other factors may be

diathermy application to the long posterior ciliary vessels, and obstruction of the retinal vessels secondary to chorioretinal scarring or compression by the tube.

12:15 p.m.

HEREDITARY ANIRIDIA
(A Motion Picture)

Olga Ferrer, M.D.
Havana, Cuba

Twenty-two members of a family of ninety individuals, representing five generations, were found to have aniridia. Some of these patients are shown and the pathologic condition demonstrated.

SECTION ON OTOLARYNGOLOGY

Thursday, October 13, 1960

2:00 p.m.

GRAND BALLROOM

Presiding Officer:
DAVID R. HIGBEE, M.D.
Vice-President

Secretary:
CLAIR M. KOS, M.D.

KODACHROME STUDIES OF TYMPANOPLASTIC SURGERY

Richard A. Buckingham, M.D.
Chicago

This kodachrome presentation will show how conservative, preservative, and restorative ear surgery can rid the patient of his ear disease and retain and improve his hearing.

The otologist can no longer be satisfied with a dry ear; by surgical intervention he must remove chronic ear disease but leave the ear an instrument of hearing.

Eardrum photographs will reveal examples of natural healing processes that Wullstein and Zollner found in their magnificent studies that led them to the discovery of modern tympanoplasties.

Preoperative and postoperative transparencies will show examples of the Wullstein types of tympanoplasty as well as the new Shea vein graft method of myringoplasty.

A preoperative and a postoperative audiogram will accompany each series of ear photographs to show the effect of surgical procedure on the hearing.

Slides will demonstrate some of the tympanoplastic techniques.

2:30 p.m.

CERVICOFACIAL CONGENITAL ANOMALIES

Richard T. Farrior, M.D.
Tampa, Florida

Discusser:
A. C. Furstenberg, M.D.
Ann Arbor, Michigan

The subject of congenital abnormalities is presented with the hope of stimulating interest in these anomalies which are so directly related to the specialty of otolaryngology. The embryology of this area is in itself fascinating, and an understanding of the anatomic development is necessary for a thorough knowledge of the surgical anatomy of the cervicofacial area. This knowledge is essential for the proper management of these cases as seen in practice.

The otolaryngologist, because of his familiarity with anatomic intricacies and the other medical and surgical diseases of this area, is most qualified for recognizing these conditions and directing their management. Should he not be interested in the actual surgical correction of the anomaly, at least he will be better able to advise his patient or the patient's parents if he is informed regarding present-day management.

This paper is illustrated with color slides correlating the developmental anatomy of the clinical management of these anomalies.

3:00 p.m.

COMBINED RADIATION AND SURGICAL THERAPY FOR CARCINOMA OF THE LARYNX

Joseph L. Goldman, M.D.
and
Sidney M. Silverstone, M.D.
BY INVITATION
New York

Discussers:

Jerome A. Hilger, M.D.
St. Paul, Minnesota
William G. Hemenway, M.D.
Chicago
BY INVITATION

3:30 p.m.

LARYNGECTOMY AND NECK DISSECTION
(A Motion Picture)

William H. Saunders, M.D.
Columbus, Ohio

3:50 p.m.

PROSTHETIC ARTICULATIONS IN STAPES SURGERY
(Includes Motion Picture)

Victor Goodhill, M.D.
Los Angeles

At the present time, endotympanic surgery for otosclerosis, pioneered by Rosen, is the preferred approach in most cases. The Lempert semicircular canal fenestration operation is still an important surgical method in far advanced oval window lesions, notwithstanding recent advocacy of radical oval window surgery.

Because of frequency of re-ankylosis in cases with crural and footplate invasion, it appears wise to bypass these areas in such cases by subtotal stapedectomy. It then becomes necessary to substitute a prosthetic transmitter from the incus to an uninvolved "virgin island" in the footplate.

It is essential for such a prosthesis to become anatomically and functionally articulated with the incus and the oval window. Recent experimental work indicates certain hazards in extensive replacement of the footplate by other tissues. An intact uninvolved footplate virgin island has been observed repeatedly in older patients with long otosclerotic histories. Therefore it seems prudent to utilize such a segment provided it can be articulated with a prosthesis. It is also essential, of course, for an articulation to exist with the incus. Such an articulation should not be left to chance (postoperative fibrous adhesions) but should be obtained at the time of operation if possible.

A motion picture will demonstrate several stapes procedures in terms of orderly surgical concepts emphasizing surgical placement of an articulated polyethylene prosthesis.

SECTION ON OPHTHALMOLOGY

Friday, October 14, 1960

9:00 a.m.

GRAND BALLROOM

Presiding Officer:
JAMES H. ALLEN, M.D.
Vice-President

Secretary:
KENNETH L. ROPER, M.D.

BILATERAL CONGENITAL GLAUCOMA
(A Clinicopathologic Case Report)

Merrill J. Reeh, M.D.
Portland, Oregon

The study of congenital glaucoma from the standpoint of histology is extremely valuable. Eyes obtained in the early course of the disease before degeneration, atrophy and fibrosis have resulted from uncontrolled, elevated, intraocular pressure offer an unusual opportunity for study. In these specimens the failure of proper cleavage, the forward attachment of the ciliary muscle to the trabecular fibers with compression of the scleral spur and the canal of Schlemm, can be demonstrated. Study of these specimens shows the canal of Schlemm to be present but unable to function properly because of the developmental anomaly. This points

out the necessity for early recognition and diagnosis followed by expertly performed goniotomy. Goniotomy must be performed while the canal of Schlemm is still present and degenerative changes are minimal or absent.

At the age of 4 months this Negro, full-term infant developed typical symptoms of congenital glaucoma. A goniotomy was performed on each eye on two occasions. The intraocular pressure remained normal for one month prior to death, which occured suddenly because of congenital heart disease and pneumonia.

9:10 a.m.

PRESENTATION OF NEW INSTRUMENTS

9:30 a.m.

MOTILITY COMPLICATIONS IN BUCKLING PROCEDURES

Hunter H. Romaine, M.D.
New York

Discusser:

Conrad Berens, M.D.
New York

The accepted procedure used in replacing muscles that have been severed at their insertion for exposure during the buckling procedure will be described and an explanation for this will be given. How the situation varies with the position of the buckle will be described. A description of the defects that occur despite efforts at prevention will be taken up. Methods of controlling or correcting defects found will be covered. An analysis of all material available will be given.

10:00 a.m.

ELECTRORETINOGRAM IN CENTRAL DEGENERATION

A. D. Ruedemann, Jr., M.D.
Detroit
and
Werner K. Noell, M.D.
Buffalo, New York

BY INVITATION

Discusser:

Jerry H. Jacobson, M.D.
New York

A series of cases illustrating various types of central retinal degeneration will be presented. These cases will include juvenile and adult central pigmental degeneration, Kuhnt-Junius disease with angioid streaks, senile macular degeneration, uveitis involving the macular area, and peripheral retinal disease progressing to the central visual area. An attempt will be made to correlate the clinical findings with the demonstrable electroretinographic changes. In general, the changes in the electroretinogram relate to variation in the form of the electroretinogram from the normal as well as alteration in the response to color stimulus and flicker. The cases presented as illustrations will be related to known normals in each instance. Several cases with unequal central visual defects will be illustrated to point out the changes described.

10:20 a.m.

REACTIONS TO DISSOCIATION AND PRIMARY INSUFFICIENCIES OF THE VERTICALLY ACTING MUSCLES

Alberto Urrets-Zavalia, Jr., M.D.
Cordoba, Argentina

Discusser:

Frank D. Costenbader, M.D.
Washington, D. C.

In cases of strabismus where, irrespective of the convergent or divergent character of the horizontal deviation, the well known phenomenon of elevation in adduction exists, a relative divergence of the visual axes is ordinarily met with in direct elevation, as is also some degree of convergence in depression. When, conversely, depression in adduction prevails, divergence in deorsumversion and convergence in sursumversion are usually encountered. That these syndromes are frequently due, respectively, to an overaction of the inferior oblique and to a primitive or inhibitional underaction of the same muscle, seems supported by a number of facts.

In some cases of long-standing dissociation of the eyes (whether occasioned by squint or by the reduced value of the retinal image in one eye) and, more rare-

ly, in persons having binocular single vision under normal conditions, innervational anomalies in the form of alternating hyperphoria, of latent nystagmus and of conjugate cycloversion are seen to appear on uniocular occlusion.

In recent years, there has been an increasing tendency to confuse the motor disturbances of this latter kind with those of the former, to which they are often associated, and to consider most instances of what obviously are genuine insufficiencies of the obliques and of the vertical recti as functional disorders.

On the basis of newly acquired evidence, the issue is discussed and the conclusion reached that in a large number of patients a true, anatomically determined disturbance of the vertically acting muscles obtains. Considerations on the diagnostic problems involved are presented.

10:50 a.m.

THERAPEUTIC LAMELLAR KERATOPLASTY WITH USE OF PRESERVED DEHYDRATED CORNEA (A Motion Picture)

A. Benedict Rizzuti, M.D.
Brooklyn

This film in color illustrates the surgical techniques employed in removing diseased cornea. The electrokeratotome is used to remove a uniform lamellar graft from a preserved dehydrated donor cornea.

11:05 a.m.

PULSATING ENOPHTHALMOS IN ASSOCIATION WITH NEUROFIBROMATA OF THE EYELID (Including a Motion Picture)

Oliver H. Dabezies, Jr., M.D.
New Orleans
and
Frank B. Walsh, M.D.
Baltimore

A 27-year-old Caucasian male had a pulsating enophthalmos in association with histologically proven neurofibromata of the left upper eyelid. At the age of 17 years he had sustained severe trauma to the left frontotemporal area. Roentgenography disclosed a defect of the orbit. The ocular pulsation was thought to be due to the cerebral pulsation being transmitted into the orbit.

Exophthalmos is the usual finding in patients with a congenital bony defect of the orbit in association with neurofibromatosis. Enophthalmos was present in our case, and was probably due to an atrophy of the orbital fat secondary to trauma.

11:30 a.m.

DURATION OF OCULOMOTOR AKINESIA OF INJECTABLE ANESTHETICS

William G. Everett, M.D.
E. Kenneth Vey, M.D.
and
James W. Finlay, M.D.
BY INVITATION
Pittsburgh

Discusser:

Daniel Snydacker, M.D.
Chicago

A new technique for evaluating the duration of motor paresis resulting from retrobulbar anesthesia which utilizes the phenomena of optokinetic nystagmus in monkeys is presented.

The authors compare the duration of extraocular muscle paresis produced by the common anesthetic agents and one new drug when injected retrobulbarly. The effect of hyaluronidase, of epinephrine, and of their combination with anesthetic drugs is also studied and discussed.

SPECIAL SCIENTIFIC PROGRAMS

These Special Programs are open meetings. All members and guests of the Academy are welcome to attend.

THE COMMITTEE FOR RESEARCH IN OTOLARYNGOLOGY
FOURTH ANNUAL MEETING
Northwestern University Medical School
303 East Chicago Avenue

Saturday, October 8, 10.00 a.m.

HANS VON LEDEN, M.D.
Program Chairman

Some Relations Between Temporary and Permanent Threshold Shifts
ARAM GLORIG
Research Center,
Subcommittee on Noise in Industry

Calculations of Arytenoid Motion
MARY ANN FRABLE
Northwestern University

Closure of the Cochlear Windows: Its Effect Upon Bone Conduction in Cats
JUERGEN TONNDORF
and
JAMES R. TABOR
University of Iowa

Neural Basis of Frequency Discrimination
JAY M. GOLDBERG
University of Chicago

Pressure Recordings in the Esophagus and Cricopharyngeus
FREDERICK L. DEY
and
JOHN A. KIRCHNER
Yale University

Habituation of Nystagmus to Caloric Tests in Cats
HILS G. HENRIKSSON
ROBERT KOHUT
and
CESAR FERNANDEZ
University of Chicago

LUNCHEON

Reconstruction of Oval Window with Homologous, Heterologous and Banked Ossicles
RICHARD J. BELLUCCI
and
DOROTHY WOLFF
New York University

Comparative Neurohistology of the Tensor Tympani and Stapedius Muscles
CHARLES E. BLEVINS
University of California
San Francisco

The Acoustic Impedance and Resonances of the Tracheo-pulmonary Apparatus
C. J. CAMPBELL
and
JOHN A. MURTAGH
Dartmouth Medical School

Electronmicroscopy of the Human Ear
WARD B. LITTON
University of Michigan

Experimental Observations on Facial Nerve Regeneration
JESSE E. WALLER
Northwestern University

Electrical Potential of the Cochlea in Response to Acoustic Transients; Cochlear Microphonics and Remote Components
DON C. TEAS
S. P. DIAMOND
and
HALLOWELL DAVIS
Central Institute for the Deaf

TEACHERS' SECTION: OPHTHALMOLOGY

Grand Ballroom

Sunday, 10:00 a.m.

A. D. Ruedemann, M.D.
Chairman

How Much Training in Research Should Be a Part of Graduate Training?

William L. Benedict, M.D.
Moderator

Alson E. Braley, M.D.
State University of Iowa College of Medicine
Iowa City

Michael J. Hogan, M.D.
University of California School of Medicine
San Francisco

A. Edward Maumenee, M.D.
Johns Hopkins University School of Medicine
Baltimore

TEACHERS' SECTION: OTOLARYNGOLOGY

Crystal Room

Sunday, 10:00 a.m.

John A. Kirchner, M.D.
Chairman

Symposium: Residency Training

Francis L. Lederer, M.D.
Moderator

I. How Should the Basic Sciences Be Taught?

Theodore E. Walsh, M.D.
Washington University School of Medicine
St. Louis

II. Should Residents Do Research?

John R. Lindsay, M.D.
University of Chicago, The School of Medicine
Chicago

III. How Much Clinical Experience Is Necessary?

Edmund P. Fowler, Jr., M.D.
Columbia University,
College of Physicians and Surgeons
New York

AMERICAN ORTHOPTIC COUNCIL
and
AMERICAN ASSOCIATION OF ORTHOPTIC TECHNICIANS

Waldorf Room
Conrad Hilton Hotel

Sunday, 8:00 p.m.

Philip Knapp, M.D.
President

Edmond L. Cooper, M.D.
Secretary

Symposium: The Nonsurgical Treatment of Strabismus

The Management of Defective Visual Acuity

William Havener, M.D.
Columbus, Ohio

The Management of the Sensory Anomalies

Barbara Simmons
Atlanta, Georgia

The Management of the Deviation

Alton V. Hallum, M.D.
Atlanta, Georgia

COMMITTEE ON CONSERVATION OF HEARING
and
OTOSCLEROSIS STUDY GROUP

Grand Ballroom

Sunday, 8:00 p.m.

DEAN M. LIERLE, M.D.
RAYMOND E. JORDAN, M.D.
Co-Chairmen

Electronic Microscopy of Cochlea

CATHERINE A. SMITH, PH.D.
St. Louis

Fluid Barrier Within the Otic Capsule

MERLE LAWRENCE, PH.D.
DAVID WOLSK, M.A.
Ann Arbor, Michigan

A Study of Hearing in Children: Objectives and Preliminary Findings

ELDON L. EAGLES, M.D., C.M., DR. P.H.
Executive Director,
Subcommittee on Hearing in Children
Pittsburgh

COMMITTEE ON RECONSTRUCTIVE PLASTIC SURGERY: OPHTHALMOLOGY

Grand Ballroom

Tuesday, 8:00 p.m.

WENDELL L. HUGHES, M.D.
Chairman

SYMPOSIUM: EXOPHTHALMOS
EDMUND B. SPAETH, M.D.
Moderator

Differential Diagnosis

JOHN W. HENDERSON, M.D.
Ann Arbor, Michigan

Treatment of Exophthalmos Associated with Thyroid Dysfunction

ROBERT M. DAY, M.D.
New York

Surgical Approach

HOWARD A. NAQUIN, M.D.
Baltimore

Management of Secondary Factors

JOHN M. MCLEAN, M.D.
New York

COMMITTEE ON RECONSTRUCTIVE PLASTIC SURGERY: OTOLARYNGOLOGY

Crystal Room

Tuesday, 8:00 p.m.

JOHN MARQUIS CONVERSE, M.D.
Chairman

The Fate of Bone Grafts in Repair of the Deformities of the Face

HARRY SICHER, M.D.
Chicago

Reconstructive Surgery for the Adult Cleft Palate Cripple

ORION H. STUTEVILLE, M.D.
Chicago

General Principles of Lateral Osteotomy

WILLIAM K. WRIGHT, M.D.
Houston, Texas

SCIENTIFIC EXHIBITS

KENNETH L. ROPER, M.D., *Chairman*
EUGENE L. DERLACKI, M.D., *Co-Chairman*
CHICAGO, ILLINOIS

LOCATION:
Registration Area, 4th Floor

Exhibit S-1

The Schiotz Tonometer—What you should know about it

THE OPHTHALMOLOGICAL FOUNDATION, INC., New York, represented by Conrad Berens, M.D., New York; R. O. Gulden, Elking Park, Pennsylvania, and Patricia Rainier, Jersey City, New Jersey

Demonstration of the various parts of the Schiotz tonometer which may not be functioning properly and thereby give a false reading. Charts, drawings, models and used instruments will be shown to demonstrate how far off standard your tonometer may be. The exhibit will be attended daily to answer questions on the care and use of the instrument. A new sterilizer for the tonometer, a bulbar compressor and bottles used for glaucoma will be shown.

Exhibit S-2

Blind People and Community Resources

AMERICAN FOUNDATION FOR THE BLIND, INC., New York, represented by Kathern F. Gruber, and Pauline M. Moor, New York, and Richard E. Hoover, M.D., Baltimore

Exhibit of pertinent literature available from the American Foundation for the Blind. Professional consultants, including an ophthalmologist, will be at the booth at all times. Information pertaining to resources within each state will be available upon request.

Exhibit S-3

Why Orthoptics?

AMERICAN ASSOCIATION OF ORTHOPTIC TECHNICIANS, represented by Helen A. Cason, Omaha, and Robert S. Miller, Milwaukee

Reasons for orthoptic therapy will be shown: (1) to achieve binocularity and (2) to improve present fusion. Some methods, simple and complex, will be demonstrated which help to accomplish these aims. Results of therapy will be presented.

Exhibit S-4

Stapes Operations in 3-D, Volume I; Tympanoplasty in 3-D, Volume II

J. BROWN FARRIOR, M.D., Tampa, Florida

The Academy has prepared, for distribution at cost, two stereoscopic atlases on ear surgery. Each atlas contains 70 stereo slides each. Each slide is accompanied by a detailed description of the photo-pathology and the surgical technique of the variable operations upon the stapes; the vein graft, the vein plug stapedioplasty and tympanoplasties type I, II, III, IV and V.

Exhibit S-5

Home Study Courses, American Academy of Ophthalmology and Otolaryngology

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, represented by Daniel Snydacker, M.D., Secretary for Home Study Courses, Chicago, and Peter N. Pastore, M.D., Associate Secretary, Richmond, Virginia

This exhibit will show the purposes, scope and organization of the Home Study Courses.

Exhibit S-6

The Seeing Eye, Inc.

THE SEEING EYE, INC., Morristown, New Jersey

Exhibit consists of a large display panel, posters, and assorted literature.

Exhibit S-7

Textbooks for the Visually Handicapped

AID TO VISUALLY HANDICAPPED, San Francisco, represented by Mr. and Mrs. Gene Marchi

The exhibit is devoted to demonstration of printed textbooks which have been transcribed into 18-point type from normal-print books used in the public schools. The work is done by volunteers of AID TO VISUALLY HANDICAPPED, INC. and distributed, free of charge to all school districts and public libraries for the express purpose of aiding partially sighted children who are unable to read books with smaller print.

Exhibit S-8

Biological Sciences 091: The Human Eye

THE HADLEY SCHOOL FOR THE BLIND and THE HOME-STUDY DEPARTMENT OF THE UNIVERSITY OF CHICAGO, Chicago, and FARRAR, STRAUS AND CUDAHY, INC., New York

Exhibit shows completion of 5-year joint project under supervision of Frank W. Newell, M.D.: A home-study course on the eye for the layman. A series of transparencies will show how this free course will benefit (1) blind professional workers for the blind, and (2) sighted students, as a public education service. Course materials will be on display: THE TRUTH ABOUT YOUR EYES by Derrick Vail, M.D., and Syllabus in print, Braille, discs, and tape, plus dissectible plastic model of the eye.

Exhibit S-9

American Board of Opticianry and Educational Foundation

AMERICAN BOARD OF OPTICIANRY, represented by Charles Sheard, Ph.D., Secretary of Board, Rochester, Minnesota, and Jack C. Copeland, Chairman Exhibit Committee, Chicago

Information regarding the functions and work of the American Board of Opticianry and its Education Foundation in Ophthalmic Optics will be made available through various printed materials and charts regarding schools, collegiate curricula, scholarships and achievement awards; classes of certification and requirements; application blanks and general information regarding character, time and places of examinations.

LOCATION:
Club Floor

Exhibit S-10

The Eye at 65 and Over

DAN M. GORDON, M.D., New York Hospital—Cornell Medical Center, New York

The commoner abnormalities which occur in the eyes of the aged are depicted. The incidence of the various eye conditions for which the geriatric patient consults an ophthalmologist will be pointed out on the basis of a breakdown of the diagnoses on a large consecutive series of patients of 65 and over. Stress is laid upon management of these conditions.

Exhibit S-11

Otologic Microsurgery: Otosclerosis Surgery and Tympanoplasty

TEMPLE UNIVERSITY SCHOOL OF MEDICINE AND HOSPITAL, Philadelphia, represented by David Myers, M.D.; Woodrow D. Schlosser, M.D.; Arnold K. Brenman, M.D.; Lindsay L. Pratt, M.D., and Richard A. Winchester, Ph.D.

Two eight-foot side panels will present color enlargements of photographs taken during tympanoplasty and otosclerosis operations. The development of stapes mobilization surgery will be traced and stages of modern stapedectomy techniques, including Shea vein graft, will be stressed.

Modern tympanoplastic techniques will be shown. A 20 × 20-inch projection screen, controlled by the narrator, will supplement the photographs with slides.

Exhibit S-12

Oncology of Head and Neck: Primary and Reconstructive Surgery

RICHARD T. FARRIOR, M.D., and GARLAND D. DUMMIT, M.D., Tampa General Hospital, Tampa, Florida

The exhibit consists of 60 enlarged color transparencies with accompanying descriptive captions, representing common and unusual lesions from each of the anatomic areas of the head and neck. Larger panels of printed material outline the general principles of cancer surgery and the advisability of primary reconstruction in selected cases.

Photographs made during surgery, representing particular techniques extending from simple out-patient procedures to major en bloc combined neck resections, are presented. Original techniques are incorporated with the currently accepted principles of cancer surgery.

The exhibit emphasizes the need for the surgeon performing major head and neck surgery to be familiar with the methods of reconstruction available both for more adequate primary excision and for the preservation of function wherever possible.

Exhibit S-13

Congenital Conditions of the Eye

EVERET H. WOOD, M.D., Lovelace Clinic, Albuquerque, New Mexico

Exhibit presents various congenital defects of the eye including anterior staphyloma, coloboma of iris, microphthalmos, congenital glaucoma, pigmented nevi, hemangioma of lid. Photographs and photomicrographs are included.

Exhibit S-14

Experimental Intraocular Venography

THE NATIONAL INSTITUTES OF HEALTH — OPHTHALMOLOGY BRANCH, NATIONAL INSTITUTE OF NEUROLOGICAL DISEASES AND BLINDNESS, represented by Bruce E. Cohan, M.D., Ann Arbor, Michigan, and Joseph M. Morel, R. T., Bethesda, Maryland

The exhibit depicts a method which permits the radiographic visualization of the intraocular venous systems in the intact cat eye. Through a small conjunctival incision, the anterior ciliary vein is cannulated. Then during perfusion with contrast substances, radiographs are taken. These roentgenograms demonstrate the intrascleral, ciliary body, and choroidal venous systems.

Exhibit S-15

Advantages of Binocular Indirect Ophthalmoscopy Demonstrated by Rubber Eye Models

MATTHEW D. DAVIS, M.D., and YVONNE MAGLI, B.S., University of Wisconsin Medical School, Madison, Wisconsin

The exhibit will consist of rubber model eyes demonstrating various impediments to adequate ophthalmoscopy by the direct method, such as lens and vitreous opacities and high myopia, and certain fundus lesions whose study is more adequate with the indirect method, such as retinal detachment, choroidal tumors, and posterior staphylomas. The ophthalmologist will examine the models by both direct and indirect ophthalmoscopy in order to compare the two methods. Scleral indentation will be demonstrated.

Exhibit S-16

Ocular Allergy

LELAND H. PREWITT, M.D., Ottumwa, Iowa

Exhibit consists of 5 × 7 and larger kodachromes of intra and extra allergies such as vasospastic retinitis, a 32-year-old physician sensitive to tobacco, showing the actual vasospasm and huge scar 6 months later; also an anaphylactic food sensitivity showing retinal edema, retinal detachment bilateral in a 15-year-old boy; an acute parasitic retinal detachment in a

14-year-old boy, as well as numerous dermatoblepharal conjunctivitis cases—arranged and mounted by the Medical Arts Department of University of Iowa Medical School.

Exhibit S-17

Surgical Treatment of Hearing Loss

MEMPHIS FOUNDATION OF OTOLOGY, represented by John J. Shea, M.D., and David F. Austin, M.D., Memphis

The surgical treatment of hearing loss as developed in the Memphis Foundation of Otology is demonstrated, including fenestration of the oval window with vein graft and polyethylene implant for otosclerosis, and vein graft closure of eardrum perforations in myringoplasty and tympanoplasty. Included also will be a description of the investigative work being done to determine the cause of sudden hearing loss, both the spontaneous type and that which occurs following surgery. A simplified method for measuring the cochlear microphonics of the ear will be shown.

Exhibit S-18

Percutaneous Renal Biopsy and Ocular Vascular Disease

THE UNIVERSITY OF CHICAGO, DEPARTMENTS OF SURGERY (OPHTHALMOLOGY), MEDICINE, AND PATHOLOGY, represented by Frank W. Newell, M.D.; John D. Arnold, M.D.; Benjamin Spargo, M.D., and Bertha A. Klien, M.D., Chicago

Renal biopsy specimens have been studied by light and electron microscopy. In some retinal vascular diseases, changes have been found in the kidney suggesting that a similar pathologic process involves both organs. In some patients with glomerulonephritis, intercapillary glomerulosclerosis (Kummelstein-Wilson) has been found associated with retinal microaneurysms prior to the onset of overt diabetes. Information concerning the indications, technique and complications of renal biopsy will be presented together with illustrative material correlating ocular and kidney disease.

Exhibit S-19

Activities and Publications of the Committee on Conservation of Hearing

COMMITTEE ON CONSERVATION OF HEARING OF THE AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

The exhibit will consist of posters depicting the activities of the Committee on Conservation of Hearing, of bibliographies of publications by the Committee, and publications by members of the Committee which are pertinent to hearing conservation. A number of the more recent publications of the Committee which can be secured through the Academy will be displayed.

Exhibit S-20

Tonography

COMMITTEE ON STANDARDIZATION OF TONOMETERS OF THE AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY, represented by Dr. Elmer J. Ballintine, Chairman, Cleveland; Bernard Becker, M.D., St. Louis; W. Morton Grant, M.D., Boston; Peter C. Kronfeld, M.D., Chicago; Robert A. Moses, M.D., St. Louis, and R. Winston Roberts, M.D., Winston-Salem, North Carolina

The exhibit will consist of five panels describing, in tables and graphs, the material presented in the glaucoma symposium. Kronfeld: The principle of tonography, terms and definitions; Ballintine: Technique of tonography, typical tonograms, calculations and interpretations; Moses: Physiologic, pathologic and instrumental variables; Grant: Experimental tonography; Becker: Tonography in the diagnosis of chronic simple glaucoma; Roberts: Tonography in the management of the primary glaucomas; Kronfeld: Summary.

Exhibit S-21

Illinois Eye Bank of the Illinois Society for the Prevention of Blindness

ILLINOIS SOCIETY FOR THE PREVENTION OF BLINDNESS, Chicago

A consultation service.

Exhibit S-22

Chamber Angle Anomalies in Developmental Glaucoma and in Systemic Mesodermal Disorders

DEPARTMENT OF OPHTHALMOLOGY AND DEPARTMENT OF ORTHOPEDIC SURGERY, COLLEGE OF MEDICINE, STATE UNIVERSITY OF IOWA, represented by Hermann M. Burian, M.D.; Lee Allen, and Ignacio V. Ponseti, M.D., Iowa City

This exhibit will consist of gonioscopic views of patients with Marfan's syndrome, idiopathic scoliosis and Legg-Perthes' disease. A high correlation was noted between chamber angle changes and their severity, and the systemic disorders and their severity. The exhibit will also include histologic presentations of the chamber angles in a case of Marfan's syndrome, and sections and reconstruction models of the eyes of a patient with developmental glaucoma.

Exhibit S-23

Maxillofacial Rehabilitation

MICHAEL L. LEWIN, M.D., and EUGENE GOTTLIEB, M.D., Montefiore Hospital, New York

This exhibit deals with congenital and acquired deformities of the jaws associated with functional disruption and cosmetic deformities. Various corrective surgical procedures in conjunction with dental restoration are demonstrated. The result provides satisfactory jaw function and restores normal esthetic appearance.

Exhibit S-24

Tympanoplasty

EDWARD CARPENTER BRANDOW, JR., M.D., and EDWARD IANDOLI, Ph.D., Albany Medical College, Albany, New York

The theory and principles of different types of tympanoplastic operations are presented. Charts outline the indications, preoperative evaluation, postoperative care and problems encountered with the operation. Diagrams demonstrate the steps. Kodachrome photographs show pathologic changes in diseased ears and the technique of the operation.

Exhibit S-25

Interference Cinemicrography of Ocular Tissues

LOUIS J. GIRARD, M.D.; CHARLES D. DUKES, Ph.D., and HILDA H. FOX, Baylor University College of Medicine, Houston, Texas

A self-contained continuous motion picture projected onto a translucent screen which can be viewed by eight to ten people at a time. Comprises time-lapse motion pictures of tissue cultures of various ocular tissues, demonstrating modes of division (mitosis), cytoplasmic activity, feeding (pinocytosis) and intracellular organization and activity.

Exhibit S-26

External Ocular Infection: A Controlled Study with Triclobisonium Chloride

UNITED HOSPITALS OF NEWARK—NEWARK EYE AND EAR INFIRMARY UNIT, represented by Richard U. Stern, M.D.; Walter T. Rados, M.D.; Humbert M. Gambacorta, M.D., and John Insabella, M.D., of Newark, New Jersey, and Herbert L. Moskowitz, M.D., of Millburn, New Jersey

One hundred twenty unselected clinic patients with external ocular infection (conjunctivitis, corneal ulcer) were treated with triclobisonium chloride, a new antimicrobial agent. Causative pathogens were cultured and identified; the predominant organism found was staphylococcus. The clinical efficacy of the drug was evaluated. Laboratory animal studies were also undertaken to extend the evaluation. Results are graphically portrayed.

Exhibit S-27

Color Television Photography of the Fundus Oculi

WALTER REED ARMY HOSPITAL AND NATIONAL INSTITUTES OF HEALTH, represented by Col. Austin Lowrey, Jr., MC, USA, and Capt. Richard B. Rich, MC, USA, Washington, D. C., and Murray C. Brown, M.D., and Willard C. Whitehouse, Bethesda, Maryland

This exhibit will display, by means of illustrative panel, the equipment and technique used in obtaining television views of the human fundus oculi. The views can be kinerecorded, affording a continuous running projection of various funduscopic views thus obtained. Televising of the human fundus oculi is a promising means of teaching the appearance and differentiation of normal and diseased ocular conditions to a large audience simultaneously. This also is a means of recording dynamic changes in the retinal vasculature.

Exhibit S-28

Facial Pain

ARNOLD P. FRIEDMAN, M.D.; CHARLES A. CARTON, M.D., and ASAO HIRANO, M.D., Montefiore Hospital, New York

The purpose of this exhibit is to present to the physician a plan for proper orientation and management of patients with the complaint of facial pain. A great part of the material represents our experiences in the diagnosis and treatment of facial pain at the Montefiore Hospital Headache Unit during the past ten years. Color drawings, tables and charts will discuss the classification, mechanism, diagnosis and treatment of this disorder, with special emphasis on "atypical neuralgias."

Exhibit S-29

Angle Glaucoma Showing Effect of Levoepinephrine

LAWRENCE L. GARNER, M.D., Milwaukee; WILLIAM W. JOHNSTONE, M.D., Chicago; ELMER J. BALLINTINE, M.D., Cleveland, and MICHAEL E. CARROLL, M.D., Chicago, representing Marquette University School of Medicine County General Hospital, Milwaukee; University of Illinois Eye and Ear Infirmary, Chicago; Western Reserve University School of Medicine, Cleveland, and Milwaukee County General Hospital, Milwaukee

A simple and practical chart reveals graphically the management and results in all types of open-angle glaucoma encountered during a 40-month period. Analysis shows the effects of Levoepinephrine 2 per cent alone or when administered combined with a miotic and/or a carbonic anhydrase inhibitor. The effects of the two new anticholinesterase agents on the most recalcitrant cases is included along with tonographic studies.

Exhibit S-30

Services of the National Society for the Prevention of Blindness

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS, New York, represented by John W. Ferree, M.D., Executive Director, and Virginia Smith Boyce, Assistant Director

Through charts, photographs and drawings, the Society's activities of principal benefit to the ophthalmologist will be described. These include a summary of statistical data on the incidence and causes of blindness, information on standards for vision testing of preschool and school children which are being used over the country, the grants-in-aid program. Screening for glaucoma, educational services for partially seeing children; eye safety and materials for use in patient education and in encouraging the public to seek regular eye care.

Exhibit S-31

Fibrosing Mastoiditis

UNITED STATES ARMY MEDICAL SERVICE, FITZSIMMONS GENERAL HOSPITAL, represented by Wendell A. Weller, Colonel, MC, Denver

Fibrosing mastoiditis is a pathological diagnosis. It may be associated with adhesive otitis media, granuloma and paresis of the facial nerve; in the more active phases, with subperiosteal perisinuous, or Bezold's abscess, meningitis, or acute osteomyelitis of the skull. A conductive hearing loss is usually present. The x-ray shows haziness or breakdown of the mastoid cells. In the antibiotic era, the diagnosis may be difficult to establish unless it is suspected. The cortical (simple) mastoidectomy is advocated in the treatment for the conservation of the middle ear structures and hearing.

TECHNICAL EXHIBITORS — 1960

Exhibitor	Booth No.
Alcon Laboratories, Inc.	58 and 59
The Alkalol Co.	29
Allergan Corporation	R-32
Ambco, Inc.	R-24
American Bifocal Company, Inc.	751
American Cyanamid Company	R-16
American Optical Co.	7 through 14
Department of Research and Development, The American Tobacco Company	R-17 and R-18
Ar-Ex Products Co.	729
The Armorlite Lens Co., Inc.	R-47
Artificial Human Eyes	R-27 and R-28
Aurex Corporation	R-20
Auros Corporation	714
J. R. Ballard, Inc.	703
Barnes-Hind Ophthalmic Products, Inc.	R-49
Barnett Optical Laboratories	37
Bausch & Lomb Optical Co.	95 through 102
Baxter Laboratories, Inc., Travenol Division	757
Rudolph Beaver	R-21
Austin Belgard RX Opticians	18 and ½ of 19
Belrose Refracting Equipment Co.	717
Beltone Hearing Aid Company	R-9
Benson Optical Company	85, 86 and 87
Bio-Technics Corporation	725
Blakiston Division, McGraw-Hill Book Company, Inc.	62
Boll & Lewis Optical Company	43 and 44
Broemmel Pharmaceuticals	63
Burroughs Wellcome & Co., Inc.	120
Burton M-D-L Division	747
Geo. H. Butterfield & Son	762
Cameron Surgical Instruments Co.	76
Chicago Reference Book Co.	R-2
ClerSite Co.	"A"
The Coca-Cola Company	26
Codman & Shurtleff, Inc.	R-41 and R-42
Contact Lens Guild, Inc.	749
Contact Lens Society of America	116
Contactisol, Inc.	735
Continental Optical Co.	R-15
Coreco Research Corp.	61
Joseph K. Dennis, Co., Inc.	51
The DeVilbiss Company	16
Dictograph Products, Inc.	765
Lester A. Dine Co.	121
Doho Chemical Corp.	34
Encyclopaedia Britannica	47
Engelhard Industries, Inc.	R-46 and ½ of R-45
Ethicon, Inc.	64
Fairfield Optical Co.	787
Grason-Stadler Company, Inc.	R-36
Grieshaber Swiss Eye Instruments	38
Grune & Stratton, Inc.	35
Guild of Prescription Opticians of America	41 and 42
Harlequin Corporation	6
Haug Drug Company	797
Paul B. Hoeber, Inc.	32
Hollister-Stier Laboratories	46
The House of Vision, Inc.	65, 66, 67 and 68
Hudson Optical Corp.	109

Exhibitor	Booth No.
Industrial Acoustics Company, Inc.	R-37
Ion Optical Company	R-11 and R-12
The Iso-Sol Co., Inc.	R-43
Fritz Jardon Laboratories	R-6
Jenkel-Davidson Optical Company	105 and 107
Keeler Optical Products, Inc.	92 and 93A
Kelley & Hueber, Inc.	R-8
Keystone View Co.	23 and 24
F. & F. Koenigkramer Co.	R-44 and ½ of R-45
Kono Sales Co., Inc.	108
Kontur Kontact Lens Company, Inc.	758
S. G. Krebs Co., Inc.	718
Lea & Febiger	53
Lederle Laboratories Division	R-10
Liberty Optical Mfg. Co., Inc.	90A
J. B. Lippincott Company	49
Little, Brown & Co.	764
P. Lorillard Company	745
Mager & Gougelman, Inc.	20 and ½ of 19
Maico Electronics, Inc.	69 and 70
Matalene Surgical Instruments Co., Inc.	81 and 82
McLeod Optical Co., Inc.	R-48
Medical Case History Bureau	115
Medical Film Guild, Ltd.	L-L
Merck Sharp & Dohme	45
Miles Reproducer Co., Inc.	755
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R. J. Reynolds Tobacco Company	27 and 28
The Rhinopto Company, Inc.	746
Richards Mfg. Co.	742
Ritter Co., Inc.	71
Roche Laboratories	719
Sandoz Pharmaceuticals	R-19
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Stanwix House, Inc.	763
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Surgical Mechanical Research, Inc.	90 B and 91
Tama, Inc.	748
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Walton Laboratories, Inc.	R-14
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Carl Zeiss, Inc.	74 and 75
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TECHNICAL EXHIBITS

The Academy is appreciative of the cooperation of all technical exhibitors.

Booths with numbers preceded by "R" are located in the Red Lacquer Room.

Booths with numbers above 700 are located on the Seventh Floor.

Alcon Laboratories, Inc., Fort Worth
Booths 58 and 59

New developments in ophthalmic and rhinologic medication resulting from continuous research exclusively in these fields may be discussed with personnel from Alcon's Research and Sales Departments. Alcon's full line of sterile ophthalmic preparations for prescriptions, office treatment and ocular surgery will be displayed, together with Alcon-efrin, a specially formulated nonstinging, nonirritating nasal decongestant in three strengths.

The Alkalol Co., Taunton, Mass.
Booth 29

The Alkalol Company is featuring Alkalol, a solution especially designed for the treatment of the mucous membranes of the nose and throat.

First compounded in 1896, its balanced formula of salts and essential oils makes a solution that is effective and at the same time nontoxic, mild and pleasant. It is alkaline and a mucus solvent.

Alkalol is an excellent vehicle for many drugs. For further information please call at Booth 29.

Allergan Corporation, Los Angeles
Booth R-32

Ambco, Inc., Los Angeles
Booth R-24

Ambco, Inc., greets you from Booth R-24, where we are showing the latest developments in fully transistorized audiometers, including five new models, a complete line of audiometers for every use and for every price.

Our line of audiometers includes battery-operated instruments as well as AC powered units. Also on display, a completely new line of transistorized speech and auditory training equipment. New portable units as well as new fixed location instruments will be exhibited.

Competent personnel will be on hand to answer your questions and offer help.

American Bifocal Company, Inc., Cleveland
Booth 751

American Bifocal Company, Incorporated, will display:

Volk Catraconoid Lenses for Aphakia; wide-angle aspheric glass lenses.

Volk Conoid Condensing Lenses for Indirect Ophthalmoscopy.

Volk Conoid Lenses for Subnormal Vision: aspheric, glass, large-diameter, clear, high-powered reading glasses for the legally blind.

Spherical Straight Top A B C Cataract Multifocals.

Spherical Straight Top A B C Occupational Trifocals.

Spherical Straight Top A B C Multifocals for general prescriptions.

American Cyanamid Company, Danbury, Conn.
Booth R-16

AMERICAN CYANAMID COMPANY SURGICAL PRODUCTS DIVISION, 1 Casper Street, Danbury, Conn., manufacturers of DAVIS & GECK SUTURES in the new Surgilope SP Sterile Strip Pack with Atraumatic Needles; VIM HYPODERMIC NEEDLES and SYRINGES. Other specialties include SURGAIRE—sterile deodorant spray ointment, TOPASIL—silicone skin protectant, CURON—elastic foam bandage, DISPOSABLE

NEEDLES AND SYRINGES, and Blood Lancets.

American Optical Co., Southbridge, Mass. Booths 7 through 14 including 7a

American Optical Company's exhibit at the Sixty-fifth Annual Meeting of the American Academy of Ophthalmology and Otolaryngology will include its Consul Fitting Room Furniture line, featuring the new Consul 44 fitting table; the Adapta-Rod Space Maker; and the five-purpose Consulette.

In lenses, Tillyer AOLITE Uniform Density lenses will be shown with Aspheric Cataract, Executive and Sovereign lenses. Also on display will be the 1961 Outdoor Eyewear line and a number of new cases.

Diagnostic instruments will include the Vistamatic Giantscope and Radiuscope. On display will be AO's new surgical microscope, and of special note, the new lensometer in three American Beauty colors and the Therm-O-Jet frame warmer.

Department of Research and Development, The American Tobacco Company, Richmond, Va. Booths R-17 and R-18

This exhibit features a full-color display depicting the growing and processing of tobacco.

Representatives from the Department of Research and Development will be in attendance and will welcome your questions on the role of research and control in the manufacture of quality cigarettes by The American Tobacco Company.

Technical literature will be available for distribution.

AR-EX Products Co., Chicago Booth 729

AR-EX Products Company will display their complete line of Hypo-allergenic Cosmetics, Dermatological Pharmaceuticals, and SAFE-SUDS, their hypo-allergenic Household Detergent. They will introduce their new clinically tested hypo-allergenic MASCARA. AR-EX representatives, competent to give you complete information regarding both new and old AR-EX Products, will be happy to welcome you at our booth.

The Armorlite Lens Co., Inc., Pasadena, Calif. Booth R-47

The Armorlite Lens Company, developers of durable hard resin lenses, will demonstrate the wide range in which light-weight, safe lenses are now available.

These will include examples of the safest of all lenses for children, athletes and industrial workers; of feather-weight aphakic bifocals, and of ultraviolet absorbing hard resin lenses. Ultraviolet absorbing lenses are crystal white and more perfectly transparent than glass to all light useful in seeing, yet they totally absorb all deleterious ultraviolets.

Artificial Human Eyes, Newark, N. J. Booths R-27 and R-28

Joint exhibitors in this booth are E. & S. Danz, New York; G. Danz & Sons, San Francisco; Richard Danz & Sons, New York; Erickson Laboratories, Seattle; Erickson Laboratories, Spokane; Greiner & Mueller, Chicago; L & B Laboratories, Inc., Fort Lauderdale; Hugo Leipold, Pittsburgh, and Earle C. Schreiber, Inc., Newark, N. J.

This exhibit of Artificial Human Eyes will include color photos of unusual cases, and a list of the services that are available to the patient and ophthalmologist. An implant and conformer for every type and preference will also be found here. We will feature a series of colored slides depicting the treatment of some of our "problem cases."

Aurex Corporation, Chicago Booth R-20

Auros Corporation, Milwaukee Booth 714

The Auros Corporation introduces the Auros Hearing Aid to the members of the American Academy of Ophthalmology and Otolaryngology. After eleven years of experimentation and local trial in the Milwaukee area, it is presented nationally to members of the medical profession.

Auros Hearing Aid is the first hearing aid built into spectacles. It is the only hearing aid made to fit an auriculostomy. With this aid, the hearing level is increased at least ten decibels more than it is with the ordinary type aid. Pictures and slides will demonstrate this aid and the steps necessary in making an auriculostomy. Slides of a patient with an auriculostomy will be shown.

J. R. Ballard, Inc., Chicago Booth 703

Barnes-Hind Ophthalmic Products, Inc., Sunnyvale, Calif. Booth R-49

Barnes-Hind Ophthalmic Products, Inc., will introduce a new concept for the delivery of sterile ophthalmic drops in sterile disposable units. A new series of drugs packaged under the trade name MINIMS for use in the ophthalmologist's office, in surgery, in first aid rooms, and for patient use will be shown, as well as a full line of contact lens solutions.

Barnett Optical Laboratories, Chicago Booth 37

Bausch & Lomb Optical Co., Rochester, N. Y. Booths 95 through 102

Modern ophthalmic instrumentation will be the core of the Bausch & Lomb technical exhibit.

Precision instruments with engineered convenience for professional men will be featured. These include the Deluxe Refracting Unit in Biscayne Blue, Green's Refractor and the Thorpe Slit Lamp with Aplanation Tonometer.

B & L's complete line of hand diagnostic instruments designed for quick, comfortable handling and scientific accuracy will also be displayed.

A staff of instrument experts will demonstrate and explain the instrument operation and purpose, and answer any questions.

The latest B & L Ophthalmic Frames and Lenses will also be on display.

Baxter Laboratories, Inc., Travenol Division, Morton Grove, Ill. Booth 757

Travenol Laboratories, Inc., will feature UREVERT, an intracranial decompressant, as a valuable agent in the reduction of intraocular pressure.

Rudolph Beaver, Waltham, Mass. Booth R-21

Austin Belgard RX Opticians, Chicago Booths 18 and ½ of 19

Beltone Hearing Aid Company, Chicago Booth R-9

The complete line of portable and desk model Beltone Audiometers for diagnostic and clinical testing will be displayed. Beltone Auditory Training Equipment and the current line of Beltone quality hearing aids will also be displayed. A Beltone factory representative will be on hand to answer any questions. A Polaroid Land camera will be given to one of the attending doctors.

Belrose Refracting Equipment Co., Chicago Booth 717

Belrose Refracting Equipment Company will display

1. Reliance equipment (new)
2. Karlo instrument tables (new)
3. Nikon vertexometer (new)
4. Used A.O. 7 B & L deluxe units
5. "Mark III ParaLathe" by George Butterfield (new)
6. Used phoroptors, projectors, units, lensometers, chairs, stands, visual training equipment, ophthalmometers, and miscellaneous equipment.

Benson Optical Company, Minneapolis Booths 85, 86 and 87

Benson Optical Company again this year will display a complete line of instruments and equipment together with the custom-built electric control center for all instruments and lights in the examining room. The latest in Hearing Aids and portable screening audiometer will be displayed. These things, together with office furniture, our planning service, contact lenses and complicated prescriptions will make Benson's booth a worthwhile visit.

Bio-Technics Corporation, Harrisburg, Pa. Booth 725

The Aeron Vaporizer represents a new standard of steam vapor therapy for sick-

room safety and convenience. The Aeron utilizes a unique new patented steam vapor device, the electrowick, which produces a steady flow of pure vapor in seconds without heating the water in the container. Nothing needs to be added to the water, which always remains at room temperature. The unit automatically shuts off when the water is used up. There is no glass shatter, no boiling over, and no scalding danger. The Aeron offers for the first time, unequaled safety, unique convenience, and positive performance.

Blakiston Division, McGraw-Hill Book Company, Inc., New York Booth 62

The Blakiston Division of the McGraw-Hill Book Company invites all attendees at the convention to visit their exhibit which will feature the following publications of special interest to ophthalmologists and otolaryngologists: SURGICAL TECHNIGRAMS by F. M. Al Akl, M.D., a concentrated presentation of surgical techniques focusing entirely on actual basic operative procedures; KERATOPLASTY by R. Townley Paton, M.D., F.A.C.S., a thorough and exactly illustrated book presenting modern techniques and data in the whole field of keratoplasty; and ATLAS OF EYE SURGERY by R. Townley Paton, M.D., F.A.C.S., Herbert M. Katzin, M.D., F.A.C.S., and illustrated by Daisy Stilwell. This outstanding work presents a new visual approach to eye surgery using 464 line drawings to show step-by step procedures for operating techniques.

Boll & Lewis Optical Company, Chicago Booths 43 and 44

The Boll & Lewis Optical Company exhibit will feature the latest developments in visual aids for patients with subnormal vision; cataract lens loaner service; contact lenses; Armorlite Plastic Lenses; domestic and foreign ophthalmic and diagnostic equipment; the latest styles in ophthalmic frames; and a new answer to comfortable ptosis frames.

Broemmel Pharmaceuticals, San Francisco Booth 63

Burroughs Wellcome & Co. (USA), Inc., Tuckahoe, N. Y. Booth 120

NEW PRODUCTS

The extensive research facilities of "B. W. & Co.," both here and in other countries, are directed to the development of improved therapeutic agents and techniques. Much basic theoretical work in our laboratories and in cooperation with internationally known institutions is contributing to the reservoir of fundamental medical knowledge.

Through such research "B. W. & Co." has made notable advances related to leukemia, malaria, diabetes, and diseases of the autonomic nervous system; and to antibiotic, muscle-relaxant, antihistaminic, and antinauseant drugs.

An informed staff at our booth will welcome the opportunity to discuss our products and latest developments with you.

Burton M-D-L Division, Santa Monica, Calif. Booth 747

Burton will feature the Harrington-Flocks Multiple Pattern Visual Field Screener, created by the Doctors Harrington and Flocks. This method of testing is a tachistoscopic presentation of extrafoveal stimuli from strategically located points within the central visual field, and an assessment of the patient's perception.

The diagnostic lights; the longwave ultraviolet 3,600-3,600 A (Wood's Light), and the Burton White Light will also be presented. These are essential for a complete ophthalmologic diagnosis. The Burton Super Power Light, floor stand model 1700, offers 1500 foot-candles of high intensity light for surgery and use in examinations. Finger tip control for rotating and angling.

Geo. H. Butterfield & Son, Portland, Ore. Room 762

Cameron Surgical Instruments Co., Chicago Booth 76

The Cameron Booth shows the latest developments in convenient instrumentation.

Cameron has further improved the Lempert Headlight—see the new rotator. Also, the safety approved current controllers—the Government standard—Suction Coagulation Electrodes, light weight Bin-

ocular Loupe with the new angle.

The Cameron true Radio Knife (pure radio frequency) does a better job in less time.

A visit at the Cameron Booth is worth your time.

Chicago Reference Book Co., Chicago
Booth R-2

ClerSite Company, Chicago **Booth A**

The Coca-Cola Company, Atlanta
Booth 26

Ice-cold Coca-Cola served through the courtesy and cooperation of the Coca-Cola Bottling Company of Chicago, Inc., and The Coca-Cola Company.

Codman & Shurtleff, Inc., Boston
Booths R-41 and R-42

Codman & Shurtleff, Inc., makers of surgeons' fine instruments since 1838, will exhibit their line of fine eye instruments, including the Beaver eye knives with detachable blades and a complete line of ear, nose and throat instruments, including special instruments for tympanoplasty, myringoplasty and stapes mobilization.

Contact Lens Guild, Inc., Rochester, N. Y.
Booth 749

Animated displays will show SPIRO-VENT CONTACT LENSES, their design, fitting and special properties. Models demonstrate how tear-flow into the spiral vents causes accelerated lens rotation (hydraulic turbine principle) to minimize "setting" and to increase lacrimal fluid circulation between lens and cornea. Additional displays show: fluorescein patterns in fitting SPIRO-VENT lenses; manufacturing methods; tinted lenses; lenses for aphakics and keratoconus cases; and the newest bifocal contact lenses. Demonstrations of modern techniques for modifying contact lenses will be given at frequent intervals on a portable adjusting machine. Instruction manuals for fitting and adjusting contact lenses will be distributed without charge.

Contact Lens Society of America, Philadelphia **Booth 116**

Contactisol, Inc., Lindenhurst, N. Y.
Booth 735

Continental Optical Co., Indianapolis
Booth R-15

Coreco Research Corp., New York
Booth 61

The Coret Camera embodies the principle of electronic flash and constant automatic control of such factors as distance, aperture, field, and exposure. Now, for the first time, Coreco offers a completely automatic professional clinical camera purposely designed to achieve the ultimate in surface, intra-oral, and intra-tubular photography. Because of the simplicity of operation, even an inexperienced doctor or nurse can achieve consistently perfect color transparencies.

Joseph K. Dennis Co., Inc., Chicago
Booth 51

The Joseph K. Dennis Company, Inc., AAOO Group Insurance Administrators have available a trained staff of Insurance Counsellors to assist Academy Members with their Group Insurance Programs. Plan to stop at booth 51 and receive your up-to-date Group Insurance Portfolio.

The DeVilbiss Company, Somerset, Pa.
Booth 16

The DeVilbiss Company cordially welcomes the members of the American Academy of Ophthalmology and Otolaryngology to visit its exhibit of Atomizers, Nebulizers, Steam Vaporizers and related Aerosol Therapy Equipment. Representatives will be pleased to discuss and answer any questions regarding the Home Use Line of Atomizers, Nebulizers and Steam Vaporizers as well as the complete Professional Line of Aerosol Therapy Equipment.

Dictograph Products, Inc., Jamaica, L. I., N. Y. **Booth 765**

Lester A. Dine Co., Levittown, N. Y.
Booth 121

Showing the very latest in clinical photographic equipment for either taking or viewing pictures. Featured in the line is the new sensational Eastman Kodak Camera. This new camera has quickly become the most popular close-up camera of its simplicity of operation and the basic low cost for the equipment. In addition we will feature the Eye Camera with the rotating electronic flashlight source and the ring light for shadowless color pictures. We also will present several new viewing systems for showing slides in the office or projecting slides for small clinics.

Doho Chemical Corp., New York
Booth 34

Doho Chemical Corporation is pleased to exhibit:

AURALGAN—Otitis media and removal of cerumen.

OTOSMOSAN—Fungicidal and bactericidal in the suppurative and aural dermatomycotic ears.

RHINALGAN—Nasal decongestant free from systemic or circulatory effect.

LARYLGAN—Throat spray and gargle for infectious and noninfectious sore throat involvements.

Mallon Chemical Corporation, Division of Doho:

RECTALGAN—For relief of pain and discomfiture in hemorrhoids, pruritus and perineal suturing.

DERMOPLAST—An Aerosol Spray for surface pain, burns and abrasions; obstetric and gynecologic use.

Encyclopaedia Britannica, Chicago
Booth 47

A great new edition of the Encyclopaedia Britannica and its correlated fact-finding and research services will be on display at this meeting.

To those who may be interested in acquiring Britannica, there is available a special exhibit discount.

Whether your interest is current or for the future, please visit our booth.

You need not invest to investigate.

Engelhard Industries, Inc., Elmhurst, L. I., N. Y.
Booths R-46 and ½ of R-45

Visitors to the Engelhard Booths (in the Red Lacquer Room) will see an excellent display of a wide variety of E. N. T. diagnostic instruments that are noted for quality, practicality and moderate cost. Some instruments on display are of recent development. Fully qualified representatives will gladly provide painstaking demonstrations of the full range of instruments, including the new DIAGNOSTOR, the desk-top controller that provides unmatched convenience for the use of ophthalmoscope, otoscope and other illuminated diagnostic aids. Ideal for all E. N. T. men.

Ethicon, Inc., Somerville, N. J.
Booth 64

Electron Sterilized surgical gut . . . stronger, more pliable, easier to handle, and new easy-open foil suture packages are featured. Exclusive Collagen Pure, Tru-Chromicized, Tru-Gauged properties of ETHICON surgical gut will be demonstrated, as well as a complete line of ATRALOC Needle Sutures for every surgical need. Tru-Permanized Surgical Silk and Surgical Cotton, along with other surgical specialties, will be shown.

Fairfield Optical Co., Mansfield, Mass.
Booth 787

The name FAIRFIELD in eyewear is like Tiffany in silverware.

In Room 787 you can relax and enjoy a review of Fairfield's recent creations in ladies' and men's impressive eyewear.

Fairfield creations are admired for their original designs but definitely demanded by the ophthalmologist because of their ease of adaption to the patient's prescription.

Grason-Stadler Company, West Concord, Mass. **Booth R-36**

Grason-Stadler Company, Inc., cordially

invite you to visit their exhibit where equipment of interest to Academy members will be displayed. Included in the exhibit will be a speech audiometer listed by the Subcommittee on Listing of Audiometers of the American Academy of Ophthalmology and Otolaryngology. A Bekesy Audiometer with unique features which facilitate administration of special tests useful in diagnosis of hearing difficulties will be displayed. Representatives of the company will be glad to discuss problems of application and operation.

Grieshaber Swiss Eye Instruments, Chicago **Booth 38**

It is our pleasure to welcome members of the American Academy of Ophthalmology and Otolaryngology to visit our display of Genuine Grieshaber Swiss Eye Instruments.

The original patterns of Greishaber instruments have maintained an exclusive reputation for excellence through the years. They are a pleasure to use and they enhance the Ophthalmic surgeon's skilled techniques.

This is your annual opportunity to inspect these fine instruments and we urge our regular customers to place their orders promptly for earliest possible delivery.

Grune & Stratton, Inc., New York **Booth 35**

Our Mr. Kurzer will be on hand to show you some of our new volumes in Ophthalmology and Otolaryngology. Included will be such books as Kestenbaum's CLINICAL METHODS OF NEURO-OPHTHALMOLOGIC EXAMINATION, 2nd edition, revised and enlarged; Guibor's SQUINT AND ALLIED CONDITIONS; Myklebust's PSYCHOLOGY OF DEAFNESS, Fox's OPHTHALMIC PLASTIC SURGERY.

Guild of Prescription Opticians of America, New York **Booths 41 and 42**

Adjustment of glasses and minor repairs will be continuously available for the convenience of Academy members and guests who may need this service. Guild materials in support of medical eye care will be on display.

Harlequin Corporation, New York **Booth 6**

Harlequin Corporation will present latest fashion eyewear featuring new seasonal colors and designs created to tie in with latest fashion trends.

Haug Drug Company, Milwaukee **Booth 797**

GLAUCON (Levo-epinephrine HCl. 2%) Ophthalmic Solution for the treatment of chronic open angle glaucoma will be featured.

Paul B. Hoeber, Inc., New York **Booth 32**

Among the new books on display will be Durham's ENCYCLOPEDIA OF MEDICAL SYNDROMES containing information about almost 1,000 syndromes including those in ophthalmology. Other books of special interest to members of the Academy will be Pack & Ariel's TREATMENT OF TUMORS OF THE HEAD AND NECK, Hollinshead's SURGICAL ANATOMY OF THE HEAD AND NECK, the Second Edition of Sugar's THE GLAUCOMAS, Ellis' MODERN TRENDS IN EAR, NOSE, AND THROAT DISEASES, and DeJong's THE NEUROLOGIC EXAMINATION. Copies of the classic work—Berliner's BIOMICROSCOPY OF THE EYE—will be on hand for your inspection.

Hollister-Stier Laboratories, Philadelphia **Booth 46**

Members of our technical service staff will be in attendance at the exhibit to discuss important special antigens and their application. Antigens of special interest to the members of the American Academy of Ophthalmology and Otolaryngology will be the mold, stinging insect antigens, bacterials, and of course pollen extracts, epidermals, and miscellaneous antigens that are so important in upper respiratory allergies.

The House of Vision, Inc., Chicago
Booths 65 through 68

The House of Vision will show a complete array of foreign and domestic equipment—some of it entirely new and different. Among other things, you will see a new bowl-type projection perimeter, a new operating light, a new group of fine English equipment and an entirely different concept in a projection focimeter.

The "900" Slit Lamp and the Aplanation Tonometer by Haag-Streit will be featured. You will also see the Zeiss line—and much of our many fine domestic lines.

Orthoptic equipment, field equipment, diagnostic equipment, refraction equipment, and safety equipment and materials will all be exhibited.

We look forward to your visit.

Hudson Optical Corp., Long Island City, N. Y. **Booth 109**

Industrial Acoustics Company, Inc., New York **Booth R-37**

Ion Optical Company, Detroit
Booths R-11 and R-12

The Iso-Sol Co., Inc., Lindenhurst, N. Y.
Booth R-43

Fritz Jardon Laboratories, Detroit
Booth R-6

The Fritz Jardon Plastic Research Corporation's display will include:

Custom artificial eyes and surgical implants

Introduction of newly developed prosthetic molding technique for eye control and positioning

Contact lens' display showing types of lenses, fitting procedures, and measuring equipment for various lenses

The booth will be staffed by technicians who will give a resume of fitting and demonstrate techniques of adjusting. Verbal and pictorial descriptions of types of lenses and their uses will be given to booth visitors.

Jenkel-Davidson Optical Company, San Francisco **Booths 105 and 107**

You are invited to inspect specialized equipment and accessories designed and manufactured by Jenkel-Davidson Optical Company. The exhibit will include the latest model of the Clinicamera, Multiple Pattern Screener, Trial Lens Cabinet, Selectachart, Electrajust and other convenient accessories for the ophthalmologist.

Keeler Optical Products, Inc., Philadelphia
Booths 92 and 93A

The FISON BINOCULAR INDIRECT OPHTHALMOSCOPE provides brilliant illumination, fully stereoscopic vision; it is lightweight and comfortable. An accessory Reflector, enabling two doctors to view simultaneously, is also valuable for teaching.

The lightweight SPOTLIGHT ATTACHMENT for the Keeler Magnifying Spectacle gives intense shadow-free illumination.

A new De Luxe Combination Set comprises the "Practitioner" Ophthalmoscope plus the WIDE-ANGLE OTOSCOPE, featuring all-nylon specula, 3.5 or 6 power telescopic magnification, ample operating space, brilliant light and low cost.

The FOREIGN BODY LOCATOR is compact and portable. It discriminates between ferrous and nonferrous metals. Highly accurate detection and localization are unaffected by other metals nearby.

Kelley & Hueber, Inc., Philadelphia
Booth R-8

We will be exhibiting our complete line of spectacle cases. We make a very wide variety in the finest of leathers, brocades and interesting novelty materials. We also will be exhibiting a wide variety of contact lens items including soaking and carrying cases, delivery packages, inventory control systems and contact lens kits. We also will show a diversified line of frame displays.

We will look forward to welcoming all our good friends and customers.

Keystone View Co., Meadville, Pa.
Booths 23 and 24

The Keystone View Company exhibit includes binocular functioning tests and orthoptic training materials. The Professional Orthoscope tests for diagnosing functional vision will be of special interest.

Orthoptic training equipment and materials for all types of cases are exhibited and will be demonstrated by competent technicians. A new testing unit for first, second, and third grade fusion, which requires no special equipment and which will enlist the eager cooperation of young patients, will be of special interest to doctors and orthoptists.

Various types of vision screening tests are available for inspection and demonstration.

F. & F. Koenigkramer Co., Cincinnati
Booths R-44 and ½ of R-45

Kono Sales Co., Inc., Charleston Hgts., S. C. **Booth 108**

We will introduce several new styles and colors in prescription eye wear. Also featured will be an exhibit of odd and interesting frames made for special cases.

Viewers who lean to the lighter side of life will be delighted with the array of gag frames which sports many newer and even daffier frames.

Kontur Kontact Lens Company, Inc., Richmond, Calif. **Booth 758**

S. G. Krebs Co. Inc., New York, N. Y.
Booth 718

A visit to our booth will convince you why even more Ophthalmologists and Otolaryngologists are using our equipment.

We will be featuring a complete line of the latest in eye surgical instruments, refracting equipment, diagnostic and visual training items, as well as a complete and varied line of specialties in the field of Ophthalmology.

Our ENT units as well as our mobilization and other ENT instruments will be of great interest to the Otolaryngologist.

We are distributors for the following manufacturers: Bausch & Lomb Optical Company, American Optical Company, Reliance, Ritter, Welch-Allyn, Sorenson, Gulden, Lawton, and many others.

Lea & Febiger, Philadelphia
Booth 53

Lederle Laboratories Division, American Cyanamid Company, Pearl River, N. Y. **Booth R-10**

Liberty Optical Mfg. Co., Inc., Newark, N. J. **Booth 90A**

J. B. Lippincott Company, Philadelphia
Booth 49

J. B. Lippincott Company presents, for your approval, a display of professional books and journals geared to the latest and most important trends in current medicine and surgery. These publications, written and edited by men active in clinical fields and teaching, are a continuation of more than 100 years of traditionally significant publishing.

Little, Brown & Co., Boston **Booth 764**

P. Lorillard Company, New York
Booth 745

We are presenting the Story of Kent Cigarettes. You will learn how Kent reduced tars and nicotine to the lowest level in its history.

In addition, you will see why Kent satisfies your appetite for a real good smoke. You will learn how Kent's natural tobaccos deliver a full, rich tobacco taste. In other words, you will discover how Kent filters best for the flavor you like.

A table cigarette box with your signature in gold will be a pleasant souvenir of your visit to the convention.

Mager & Gougelman, Inc., Chicago
Booths 20 and ½ of 19

Complete Artificial Eye Service, Facial Restorations and Precorneal Lenses. Our skilled glass and plastic eyemakers are heirs to our 107 years of experience. We have offices in many principal cities, and we visit other localities regularly for the fitting of artificial eyes.

We provide extensive mail order service for doctors who must do their own fittings. FREE COLOR CHART AND SIZE GUIDE available at our booth, where you are also invited to see our implants, conformers, artificial eyes, anatomical eye models, facial restorations and precorneal lenses.

We will welcome the opportunity of discussing with you any artificial eye or lens problems you may have.

Maico Electronics, Inc., Minneapolis
Booths 69 and 70

Matalene Surgical Instruments Co., Inc., New York **Booths 81 and 82**

Matalene Surgical Instruments Co., Inc., will exhibit an improved binocular gonioscope with coaxial illumination, the Chamlin perimeter, tonometers, trial frames, and the newest developments in forceps scissors, cutting instruments, instrument cases, etc., as well as the standard patterns.

McLeod Optical Company, Inc., Providence, R. I. **Booth R-48**

Medical Case History Bureau, New York
Booth 115

Simplifying the Doctor's History Record and Bookkeeping System with the INFO-DEX RECORD CONTROL SYSTEM.

Maintenance of accurate, informative data on both history and financial records is essential in the modern doctor's practice. The INFO-DEX Record Control System helps to keep a constant finger on the physical and financial status of the patient. This system correlates information almost automatically for instant reference and research work. The method of cross-indexing interesting cases according to the disease is unique and exclusive.

The MEDICAL CASE HISTORY BUREAU of New York City has specialized for many years in record forms for the doctor's office. Its well-informed representative will gladly demonstrate the INFO-DEX System and discuss your office problems.

Medical Film Guild, Ltd., New York
Booth L-L

Merck Sharpe & Dohme, Philadelphia
Booth 45

A new adrenocortical steroid is featured at the Merck Sharp & Dohme booth. 'DECADRON' dexamethasone possesses all the basic actions and effects of other glucocorticoids but in different degree. Its anti-inflammatory activity is more potent on a weight basis than any other known glucocorticoid. Electrolyte imbalance is not ordinarily a therapeutic problem.

'HydroDIURIL', a new, orally effective, nonmercurial diuretic-antihypertensive agent is also of interest. This compound is the most potent diuretic agent presently available, equaling or exceeding even the most potent parenteral organomercurials in diuretic activity.

Technically trained personnel will be present to discuss these and other subjects of clinical interest.

Miles Reproducer Co., Inc., New York
Booth 755

Newest "Walkie-Recordall" self-powered briefcase recorder-reproducer records at a radius of sixty feet minimizing background noises. No wires—no plugs. Ideal for the recording of lectures, conferences, staff meetings, case histories, provision for dictating under noisiest conditions in car or plane. Possible to record without supervision. While facilities for transcription are available, transcribing may be eliminated because the recordings are nonmagnetic, hence permanent and cost only as low as three cents per hour. They are indexed, mailable, and fileable. Featherweight file holds up to 100 belts—each to three or eight hours—can be carried in pocket.

Unique "Voice-Actuator" permits the recording automatically and without supervision as soon as voice starts. Recorder stops automatically approximately four to five seconds after voice ceases. Likewise for telephone conversation automatic recording.

World's only recorder of its kind.

Miller Surgical Co., Chicago Booth 89

See the Miller Electrosurgical units with Snares, Suction-Coagulation attachments and other special accessories for eye, ear, nose and throat procedures. These cutting, coagulating, and desiccating units, with or without spark gap, are calibrated to do the most delicate work as well as light major surgery of the eye, ear, nose and throat. There will also be on display the illuminated otoscope, ophthalmoscope, eyespud with magnet, transilluminating lamps, reflecting headlite and variable wall Rayostat which converts battery operated equipment to electric.

Modern Optics, Inc., Houston Booths 72 and 73

The C. V. Mosby Company, St. Louis Booth 88

New knowledge, new ideas, new research and technique—all are waiting for you in the newest Mosby books for 1959 and 1960. Come in. Look over these books at your leisure and convenience. If you wish his assistance, our experienced representative will be happy to discuss any book with you.

V. Mueller & Company, Chicago Booths 1 through 5

The new Michelson-Sequoia Air Drill, for stapes mobilization and related endaural procedures, will be a major feature of V. Mueller & Company's large display of instruments and equipment for this type of surgery. The Mueller Zeiss (Shambaugh-Derlacki) Operating Microscope, will be another major feature demonstrated. The improved Model MC Electronic Tonometer and the new dual Model TR Electronic Tonographer will further highlight an interesting and practical collection of fine standard and special instruments for all eye, ear, nose and throat work. V. Mueller instrument specialists will welcome your instrument problems and suggestions.

Mueller Welt Contact Lenses, Inc., Chicago Booths R-39, R-40 and 760

Mueller Welt, Chicago, will feature the most successful contact lens development by our research in recent years—the TRICON lens. This lens has three distinct curves, a central base curve fitted parallel to the flatter corneal meridian so that no central corneal touch exists, and two additional flatter curves at the lens periphery to assure adequate lacrimal circulation. The junctions between the curves are well blended to ensure smooth transitional zones and eliminate eye-sensation. The success of this lens lies in the relationship of the peripheral curves to the base curve and to each other.

Twice daily, contact lens' demonstrations will be held in Room 760.

New Era Optical Company, Chicago Booths R-3 and R-4

Obrig Laboratories, Inc., New York Booth 110

The Obrig Laboratories will present the various types of contact lenses as well as diagnostic and gonioscopic lenses.

Literature and reprints of articles will be available. Arrangements may be made for instruction in the fitting of contact lenses.

Omega Instrument Co., New York Booth 118

Ophthalmos, Inc., New York Booth R-13

You are cordially invited to visit our exhibit booth.

We will be featuring—

The STERIDROPPA in its new container.

The STERIDROPPA is a sterile single

dose disposable unit containing Ophthalmos solutions for surgery. This new container is made from revolutionary plastic material—clear as crystal—electronically sealed—absolutely impermeable and unaffected by liquids and gases.

We will also feature—

ALMOCARPINE — Ophthalmos solution of Pilocarpine Hydrochloride

EPITRATE—Epinephrine Bitartrate 2%

VAS I ZINC—Zinc Sulfate ¼%

METHISOL—Methylcellulose 4000 cps. ½%

FLUOR I STRIP—sterile disposable single dose fluorescein applicators

Our staff of trained representatives will be available to discuss with you the uses of these preparations in addition to other products for ophthalmic therapy and diagnosis.

Optical Import Co., Bellingham, Wash. Booth 756

Optical Import Company will display the latest imported ophthalmic instruments and equipment. This will include the latest developments in Pleoptics. Optical Import Company has made a large collection of translated German technical papers as well as English and American articles on Pleoptics. These are available without charge. The Pleoptics display includes the visuscope, euthoscope, coordinator and flashing controller. Optical Import Company manufactures and imports the finest opthalmic instruments and equipment.

The Opticase Company, Newark, N. J. Booth 15

Our exhibit this year will feature, as usual, a very wide range of eyeglass cases and frame display kits. We will also show for the first time something new in magnifying glasses.

Otarion Listener Corporation, Ossining, N. Y. Booth 761

Otarion Listener Corporation demonstrates the advantages of Direct Frontal Hearing—achieved by a minute microphone in front frame of eyeglasses. On display by the manufacturers of the original hearing aid built into eyeglasses is a complete line of Listener eyeglass hearing aids, including bilateral, binaural, bone conduction, single temple and telephone pickup types. Other features are tiny at-the-ear instruments and conventional aids and demonstration of the "one unit" combination puretone and speech audiometer.

Parsons Optical Laboratories, San Francisco Booths R-25 and R-26

Parsons Optical Laboratories, the world's largest manufacturers of glaucoma equipment, will show (1) both diagnostic and surgical instruments in that field, including the Barkan-Koeppe lens and the Barkan goniotomy lens; (2) floor model gonioscopes, hand-held microscopes, the Barkan focal illuminator and operating light, and a complete display of tonometers; (3) other items of our exclusive manufacture, such as the Costenbader accommodometer and the Costenbader adjustable clip-on trial frame, Harrington pocket charts, Engel fundus contact lens, Thorpe fundus lens, and Thorpe x-ray localization lens; (4) some equipment by Haag-Streit, whom Parsons represents as West Coast distributors.

George P. Pilling & Son Company, Philadelphia Booths 77, 78 and 79

The Pilling Company display will be a selection of quality instruments for Bronchoesophagology, both routine instruments and new innovations. Of particular interest will be the popular line of new Chevalier L. Jackson designs. The improved Fotobronchoscopic equipment with greater lighting system will also be on display.

A new Anterior Commissure Laryngoscope by Dr. Paul H. Holinger will be featured.

Plastic trachea tubes will be on display.

We will also show a complete line of Eye Instruments with special emphasis on the Pilling Tonometer designed by R. O. Gulden, conforming to the specifications of the Committee on Tonometers.

Plastic Contact Lens Co., Chicago Booth R-1

A complete display of all types of contact lenses, including those specifically designed for the fitting of keratoconus, aphakia and subnormal vision. Professionally accepted SPHERCON as well as BICON (bifocal contact lenses) will also be shown. In addition, all contact lens instrumentation and supplies will be available.

Alfred P. Poll, New York Booth 119

Precision-Cosmet Co., Inc., Minneapolis Booths R-30 and R-31

W. F. Prior Company, Inc., Hagerstown, Md. Booth 106

A UNIQUE AND INDISPENSABLE SERVICE FOR THE OTOLARYNGOLOGIST

In addition to the LOOSE-LEAF OTOLARYNGOLOGY and the vast number of new and revised chapters that have already been published for these volumes, you will see on display the unique OTOLARYNGOLOGY RESEARCH SERVICE. The actual requests from subscribers, the response by way of literature supplied, and the reactions of the individual subscribers will prove how valuable this service would be to you.

Gone are the days when it was necessary for the busy specialist to spend valuable time hunting for needed references. By merely mailing a request card, the literature completely answering your question is placed on your desk.

This service is vast in its comprehensiveness and negligible in cost.

Professional Pharmacal Company, Inc., San Antonio, Texas Booth R-33

The Professional Pharmacal Company will display its complete line of BufOpto Ophthalmic Solutions.

Three new BufOpto Ophthalmic Solutions will be featured. BufOpto NEO-FLO, an Ophthalmic Irrigating Solution in the new Bellows Bottle will be shown. This solution is ideal for office irrigating procedures after tonometry, gonioscopy, removal of foreign bodies, and use of Fluorescein.

BufOpto CARBACEL ¼ per cent & 1½ per cent, a new "double action" Miotic for use in glaucoma.

BufOpto MYTRATE, a sterile Methulose solution containing 1-Epinephrine Bitartrate 2 per cent for effective management of uncontrolled chronic simple glaucoma.

Our special representatives will be on hand to answer all questions concerning these products.

The Professional Press, Inc., Chicago Booth 52

The Eye, Ear, Nose and Throat Monthly is now in its thirty-ninth year of publication. Its constant aim is to provide profitable reading of a practical nature for those engaged in the eye, ear, nose and throat field.

Purdue Frederick Company, New York Booth 704

Radium Chemical Company, Inc., New York Booth 30

Radium Chemical Company, Inc., will display the Monel metal radium nasopharyngeal applicator, used for the treatment of hyperplastic lymphoid tissue at the orifice of the eustachian tube and the fossa of Rosenmueller. A complete line of instruments for the application of radon will also be shown.

R. J. Reynolds Tobacco Company, Winston- Salem, N. C. Booths 27 and 28

Welcome to the R. J. Reynolds Tobacco Company Exhibit! You are cordially invited to receive a cigarette case (monogrammed with your initials) containing your choice of CAMEL, WINSTON Filter, Menthol Fresh SALEM, or CAVALIER King Size Cigarettes.

The Rhinopto Company, Inc., Dallas Booth 746

The Rhinopto Company cordially invites physicians to visit its booth. Featured will be Rhinall Nose Drops and Spray, a time-

proven, safe nasal decongestant without burning or irritation for infants, children and adults; and Rhinall Cough Syrup for effective relief of cough and congestion.

Richards Mfg. Co., Memphis Booth 742

Ritter Co., Inc., Rochester, N. Y.
Booth 71

Ritter Equipment enables the ear, nose and throat specialist to treat **more** patients **more** thoroughly with **less** effort in **less** time.

Featuring the ever popular Ritter Ear, Nose & Throat Treatment Unit, which places all of the essential electrical, vacuum and pressure treatment facilities instantly right at your finger tips.

The NEW Ritter Motor Chair, providing greater patient comfort and a full 18 inch effortless elevation from extreme low to high position; also a special motor-elevated Chair-Table designed especially for eye, ear, nose and throat work.

Plan to spend some time at the Ritter Exhibit, and let us explain the many benefits to be derived from a lifetime investment in efficient Ritter equipment.

Roche Laboratories, Nutley, N. J.
Booth 719

Sandoz Pharmaceuticals, Hanover, N. J.
Booth R-19

Sandoz Pharmaceuticals cordially invites you to visit our display.

MALLARIL—first potent tranquilizer with a selective action (i.e.—no action on vomiting centers). This unique action gives specific psychic relaxation with safety at all dosage levels.

CAFERGOT PB—the most effective oral medication for the relief of migraine headache with gastrointestinal tract disturbance accompanied by tension.

PLEXONAL—preferred daytime sedative-relaxant. Superior to both the barbiturates and Meprobamates.

Any of our representatives in attendance will gladly answer questions about these and other Sandoz products.

W. B. Saunders Company, Philadelphia
Booth R-35

Saunders is proud to be the publisher of the new OPHTHALMIC ATLAS OF PATHOLOGY under the joint sponsorship of the Armed Forces Institute of Pathology and the Academy. It is our feature this year.

Schering Corporation, Bloomfield, N. J.
Booth 33

A cordial invitation is extended to the members of the American Academy of Ophthalmology and Otolaryngology to visit the Schering exhibit. The entire exhibit will be devoted to the use of the cortico-steroids—METIMYD (Corticoid-Antibacterial combination), METICORTEN and DERONIL—in topical and systemic therapy of allergic and inflammatory diseases of the eye. Extensive clinical and laboratory data demonstrating certain advantages of these steroids over cortisone and hydrocortisone will be presented.

Schieffelin & Co., New York Booth 50

SCHIEFFELIN & CO.'s professional representatives are exhibiting an assortment of ophthalmologic products of interest to the attending physician.

Of special interest are CYCLOSONE, a new anti-inflammatory and mydriatic agent, and CHYMEXON, a new zonulytic enzyme for cataract surgery.

Also on exhibit is CYCLOGYL, "the drug of choice" for routine refractions. CYCLOMYDRIL, for fast-acting mydriasis, and CYCLOBAR, for cycloplegic, mydriatic and antibiotic action, are also displayed.

The professional representatives will be available for discussion and service.

Robert B. Scott, Chicago Booth R-34

Shuron Optical Company, Geneva, N. Y.
Booths 83 and 84

The Shuron Optical Company Division of Textron Inc. will display its complete line of quality frames and cases. Its representatives will be glad to explain and

demonstrate the importance of correctly designed and constructed ophthalmic frames, and why special lenses for occupational and sports wear should be prescribed by the doctor. Included will be the combination Ronsir and Rondon Browline frames—both in zylonite and high purity aluminum, the all zyl Rondean in solid colors and clear bridge, Widesite Corrected Curve lenses in White, Tonetex, Greentex, Neutrex and DS (Dress Safety), and the newest in spectacle cases.

Smith-Dorsey Pharmaceuticals, Lincoln, Nebraska **Booth 741**

You and your family are cordially invited to visit the Smith-Dorsey room. Medical Service Representatives will be on hand to acquaint you with several of the newer preparations available with distinct application in your specialty.

Smith, Kline & French Laboratories, Philadelphia **Booth R-29**

Our representatives cordially invite you to discuss with them: (1) 'Ornade' Spansule® capsules, the unique oral nasal decongestant that contains a special drying agent in addition to a decongestant and an antihistamine; (2) Temaril® Tablets, Syrup and Spansule® capsules, the oral medication specifically for the relief of itching; (3) Teldrin® Spansule® capsules, for rapid and sustained antihistaminic effect; (4) Vasocort® 'Spraypak'—hydrocortisone and two decongestants to reduce intranasal inflammation and congestion; and (5) Trisocort® 'Spraypak'—hydrocortisone, three antibacterials and two decongestants for intranasal infections.

Smith, Miller & Patch, Inc., New Brunswick, N. J. **Booth R-22**

Smith, Miller & Patch, Incorporated, features LIPOTRIAD—gratifying response in senile macular degeneration, diabetic and arteriosclerotic retinopathies. A potent combination of lipotropes and oxytropic B-complex factors, in liquid or capsule form.

BISTRIMATE—first choice in "chronic sore throat." Recent reports on 395 patients with chronic sore throat treated with BISTRIMATE indicated 89.1 per cent complete or partial relief.

Solex Laboratories, Inc., Los Angeles **Booth 730**

We will display a panorama of corneal lens manufacturing and fitting techniques. We will also show the latest machinery in beveling equipment.

Perhaps the most significant part of our exhibit will be the showing of the Tuohy Ten and Tuohy Twenty corneal lenses. These new lens designs represent quite an improvement in our regular design in that the inside surface of the lens is microscopically textured throughout to present different surface ratios to the corneal surface of the eye, during eye movements, thus reducing the adhesion of lens to cornea.

Sonotone Corporation, Elmsford, N. Y. **Booth 80**

E. R. Squibb & Sons, New York **Booth R-38**

E. R. Squibb & Sons has long been a leader in the development of new therapeutic agents for prevention and treatment of disease. The results of our diligent research are available to the medical profession in new products or improvements in products already marketed.

At booth R-38, we are pleased to present up-to-date information on these advances for your consideration.

Stanwix House, Inc., Pittsburgh **Booth 763**

The "Large Type Edition" publications available from Stanwix House, Inc., are ink-print materials designed and printed in extra large type for readers with low vision. They are contemporary models of special publications having a 40-year history. Such books were first made available in 1920 by the Clear Type Publishing Committee, headed by the late Dr. Robert B. Irwin, Executive Director, American Foundation for the Blind. In the succeeding Stanwix House program, continual adherence to needs, research, and develop-

ment has resulted in quality texts, workbooks, and literary works which enhance rapid, accurate reading by the visually handicapped.

Storck Pharmaceuticals, Inc., St. Louis, Mo. Booth 25

METALEX—basic in the care of the aging—combines pentylenetetrazol and nicotinic acid in elixir and tablet forms for relief of tinnitus and loss of auditory acuity, range and amplitude. Also, in visual defects due to retinal arteriosclerosis.

PENTERGOT — provides symptomatic relief in migraine, tension, histaminic cephalalgia and vascular headaches by using a synergistic combination of caffeine and ergotamine tartrate for cerebral vasoconstriction.

ECTASULE—provides sustained, long action, uniform bronchodilation and nasal decongestion for twelve hours with one dose. Patient has benefit of complete nocturnal protection, increased exercise tolerance and increased vital capacity in allergic disorders.

Storz Instrument Company, St. Louis Booths 111 through 114

A display of fine surgical instruments including all new instruments developed during 1960 as well as all of the standard patterns.

The otologists will have an opportunity to make a comparison of all available stapes drills; speed reduction devices for regular electric drills, battery-powered drills, air-powered drills and a revolutionary new air-powered burr.

The Zeiss surgical microscope with all available observation and photographic accessories will be demonstrated.

See the Fuchs operating stool. A new design featuring cyclists seat, formed back rest, hydraulic elevation adjustable by the surgeon while seated.

Surgical Mechanical Research, Inc., Los Angeles Booths 90B and 91

A new and interesting eye cabinet.

Surgical Mechanical Research will display an efficiently and conveniently arranged office, economical in use of available space, and reasonable in cost, which will maintain its appearance of newness over the years.

See the new J-6 series treatment cabinets in both wall and floor models, featuring a pull-out writing shelf and divided drawers on heavy duty nylon rollers and glides. Drawers can be removed from cabinet.

Other treatment units, treatment chairs, stools, cuspidors, suction and pressure apparatus, tables, lights, and other sundry treatment room equipment will be shown.

We will also show the SMR EAR STOPPER, models 506 and 600, used extensively for the prevention of noise-induced hearing loss. They are equally effective for occluding the ear canal for other purposes. We will welcome questions relating to ear protection use, fitting, care, cleaning, etc.

Tama, Inc., Chicago Booth 748

The Thermosector Company, Oak Park, Ill. Booth 31

The Thermosector, an electrosurgical unit expressly designed for eye, ear, nose, and throat techniques in office and hospital, will be exhibited and demonstrated. You are invited to investigate this device and actually see its superior performance.

See, also, the Dazor Floating Magnifier Examining Lamps which will be displayed and note how they can help you and your patients on prescription.

A line of Selsi Prismatic Binoculars and Visual Aids will also be offered. They should interest you.

Titmus Optical Co., Inc., Petersburg, Va. Booth R-23

Uhlemann Optical Company, Chicago Booths 54 through 57

The Univis Lens Company, Dayton, Ohio Booths 39 and 40

Victory Optical Mfg. Co., Newark, N. J.
Booths 103 and 104

The latest exhibit booths of Victory Optical Manufacturing Company will feature "the styles of tomorrow today," and the latest colors in fashion eyewear. All Victory frames are precision crafted and ophthalmically correct, exemplifying the most desirable features of fine fashion eyewear. The best quality and the fastest service is the password in every department.

Vision-Ease Corp., St. Cloud, Minn.
Booth 60

The Vision-Ease Corporation will display its full line of improved multifocals with special emphasis on the Catarex service, which includes the Catarex temporary and Catarex permanent lenticular lens forms. On display will be the Slide Rule Adaptation of "Ranges of Usable Clear Vision Chart for Multifocal Lenses" and a new attractive absorptive tint lens holder suitable for use by the ophthalmologist in discussing absorptive tints with his patients.

Dr. Max M. von Stirne, Philadelphia
Booth R-7

Walton Laboratories, Inc., Irvington, N. J.
Booth R-14

Wampole Laboratories, Stamford, Conn.
Booth 720

Wampole Laboratories will feature, ORGANIDIN, the unique organically bound iodine useful as a mucolytic and expectorant without the side effects normally seen in conjunction with iodide therapy.

VoSol OTIC SOLUTION, for the treatment and prevention of Otitis Externa. VoSol is bactericidal and fungicidal and contains no antibiotics or steroids and causes no sensitization.

Warner-Chilcott Laboratories, Morris Plains, N. J.
Booth R-5

Welch Allyn, Inc., Skaneateles Falls, N. Y.
Booth 17

White-Haines Optical Co., Columbus, Ohio
Booths 21 and 22

The Williams & Wilkins Company, Baltimore
Booth 36

Winthrop Laboratories, New York
Booth 117

Carl Zeiss, Inc., New York
Booths 74 and 75

Carl Zeiss, Oberkochen Fundus Camera with electronic flash and Carl Zeiss, Oberkochen Operation Microscope with Photo Attachment will be again featured in our exhibit. Furthermore, the well-known Carl Zeiss, Oberkochen Slit Lamps on instrument table and compound stage, as well as accessories such as anterior chamber depth micrometer, photo attachment and gonioscopic accessories will also be displayed. In addition, the Twin Lamp, Hand Lamp, Diagnostic Set, Vertex Refractionometer and various laboratory microscopes will also be shown.

Zenith Radio Corporation, Chicago
Booths 93B and 94

The Hearing Aid Division of Zenith Radio Corporation will exhibit its highest quality, precision built audiometer. Zenith's complete line of highest quality low-cost all-transistor hearing aids will also be on display, and Zenith representatives will be on hand to demonstrate them and to explain how any physician may test a Zenith hearing aid for thirty days at no cost or obligation.

Zylite Products Co., Inc., Long Island City, N. Y.
Booth 48

Zylite Products Co., Inc., will display their 1960 collection of the latest in frame fashions.

Prominently featured will be CIRCA-LITE and SARENA-LITE, representing Zylite's first contribution to the gold-filled aluminum combination frame field. Both of these frame fashions represent a distinct contribution to the styling of combination frames which will appeal to women of all ages.

On hand to greet their many friends at the Academy Meeting will be Herman Lieberman, Charles Ladew, Peter Foy, Jack Hinkes and Homer Millikan.

SECTION ON INSTRUCTION

* * *

SIXTY-FIFTH ANNUAL MEETING

American Academy of Ophthalmology and Otolaryngology

October 9-14, 1960

PALMER HOUSE

Chicago, Illinois

A. D. RUEDEMANN, M.D.
Secretary for Instruction — Ophthalmology

EUGENE L. DERLACKI, M.D.
Secretary for Instruction — Otolaryngology and Maxillofacial Surgery

The following condensed schedules of instruction courses are reprinted for the convenience of Fellows who have not yet placed orders for tickets.

Courses already sold out are so designated on the charts. Please do not request tickets for these courses.

For complete listing of course titles, instructors, and descriptive paragraphs about each subject, please refer to the May-June issue of the TRANSACTIONS.

An *individual* course is complete in one (1) period. A *continuous* course runs two (2) or more periods as indicated. It is not possible to purchase a ticket to only *part* of a continuous course. Some courses are repeated on different days allowing the subscriber to choose the time when he wishes to attend.

Ordering Instruction Tickets

Only members who have paid their 1960 dues may order instruction tickets in advance of the meeting. **Members should order tickets only for their own use.** *Candidates for fellowship in the Academy are not privileged to reserve tickets in advance.* After the session opens, no distinction between members, candidates and guests will be made in selling tickets.

At the end of this section is printed a colored sheet to be used when ordering tickets in advance. Specify preferences on this sheet and mail to the executive office. Careful attention to instructions will ensure the correct filling of your requests. *Since number of tickets for each course is limited, be sure to list second and third choices for each period in case first choice is already sold out. In every case, effort will be made to furnish first choices.*

If you are requesting registration in the continuous courses, please specify preferences for individual courses as substitutes in order to avoid disappointment should the continuous courses which you select be sold out when your order is received.

Each period (1 hour) of instruction costs $2.00. *Orders not accompanied by properly executed checks will not be filled.* Checks should be made payable to W. L. Benedict, M.D., and mailed to the Academy office, 15 Second St. S.W., Rochester, Minn.

All orders for tickets will be filled according to the postmark on the envelope containing the check. Telegrams will not be honored. Orders will not be accepted for presale after September 29; to ensure acceptance they should be mailed before September 27.

Acknowledgment of Advance Order

As soon as an advance order is filled, a post card, listing the courses that have been reserved for him, will be mailed to the subscriber. Please do not ask to change courses after your order has been filled. The post card is merely an assurance that your order has been filled; it is not necessary for registration at the Academy meeting.

Claiming Instruction Tickets

No tickets will be sent through the mails. Members who have ordered instruction course tickets will register at the *Advance Order Section* of the Registration Desk during the week of the Convention to secure registration badge, tickets, and all convention material.

Tickets will be on sale during the meeting at the Instruction Desk in the Registration Foyer and the Seventh Floor.

Refunds

If courses requested are not available and it is impossible to substitute, a refund will be granted. A blue refund card will be placed in the Advance Registration Envelope which is received at the time of registration. This may be presented at the Instruction Desk for payment.

In case of inability to attend the meeting after order has been filled, a refund will be granted if requested prior to October 7.

If you have ordered tickets in advance, at the time of registration take your registration card to the desk marked ADVANCE ORDERS. There you will be given an envelope containing badge, tickets and program which has been prepared ahead of time to facilitate your registration.

KEY TO DESIGNATION OF PERIODS

The periods will be designated with the following key: Select tickets for — Otolaryngology, forenoons, periods 1-2-3 — Ophthalmology, afternoons, periods 4-5-6

Day	(M)onday	(T)uesday	(W)ednesday	(Th)ursday
Period a.m.		1 2 3	1 2 3	1 2 3
Period p.m.	4 5 6	4 5 6	4 5 6	4 5 6

M-4	2:00 p.m. to 3:00 p.m.	W-4	2:00 p.m. to 3:00 p.m.
M-5	3:15 p.m. to 4:15 p.m.	W-5	3:15 p.m. to 4:15 p.m.
M-6	4:30 p.m. to 5:30 p.m.	W-6	4:30 p.m. to 5:30 p.m.
T-1	9:00 a.m. to 10:00 a.m.	Th-1	9:00 a.m. to 10:00 a.m.
T-2	10:15 a.m. to 11:15 a.m.	Th-2	10:15 a.m. to 11:15 a.m.
T-3	11:30 a.m. to 12:30 p.m.	Th-3	11:30 a.m. to 12:30 p.m.
T-4	2:00 p.m. to 3:00 p.m.	Th-4	2:00 p.m. to 3:00 p.m.
T-5	3:15 p.m. to 4:15 p.m.	Th-5	3:15 p.m. to 4:15 p.m.
T-6	4:30 p.m. to 5:30 p.m.	Th-6	4:30 p.m. to 5:30 p.m.
W-1	9:00 a.m. to 10:00 a.m.		
W-2	10:15 a.m. to 11:15 a.m.		
W-3	11:30 a.m. to 12:30 p.m.		

(Familiarity with this key will be helpful and will avoid confusion)

The Following are Condensed Schedules of Courses Previously Described

CONTINUOUS COURSES — OPHTHALMOLOGY

COURSE NO.	SPEAKER	SUBJECT	PERIODS
1	Allen et al.	Bacteriology & Infectious Diseases	8 PERIODS M-4, M-5, T-4, T-5 W-4, W-5, Th-4, Th-5
2	Ascher	Aqueous Veins in Glaucoma	2 PERIODS M-4, M-5
3	Ballintine & Purnell	.....SOLD OUT.....	
4	Becker & Kinsey	Studies of Aqueous Flow	2 PERIODS T-4, T-5
5	Bedell	Ophthalmoscopy	3 PERIODS M-4, T-4, W-4
6	Berliner	Recent Advances in Biomicroscopy	3 PERIODS M-6, T-6, W-6
7	Boeder	Practical Optics	2 PERIODS Th-4, Th-5
8	Braley	Fundus Lesions	2 PERIODS W-5, W-6
9	Brockhurst & Regan, C.	.....SOLD OUT.....	
10	Brown et al.	Eye Muscles	8 PERIODS M-4, M-5, T-4, T-5 W-4, W-5, Th-4, Th-5
11	Byrnes	Ophthalmic Filters	2 PERIODS T-5, T-6
12	Callahan et al.	Ophthalmic Plastic Surgery: Advanced	8 PERIODS M-5, M-6, T-5, T-6 W-5, W-6, Th-5, Th-6
13	Clark, G.	Retinal Detachment	3 PERIODS M-6, T-6, W-6
14	Constantine et al.	Contact Lenses	6 PERIODS T-4, T-5, W-4, W-5 Th-4, Th-5
15 or 16	Copeland	Streak Retinoscopy	4 PERIODS M-4, M-5, T-4, T-5 or W-4, W-5, Th-4, Th-5
17	Costenbader et al.	Orthoptics for the Practicing Ophthalmologist	3 PERIODS M-4, M-5, M-6
18	Fonda & Sloan	Subnormal Vision	2 PERIODS T-4, T-5
19	Gibbons & Cunningham	Educational Management of Partially Seeing Children	2 PERIODS T-4, T-5
20	Gibson et al.	Surgery of Heterotropia	4 PERIODS W-5, W-6, Th-4, Th-5
21	Hughes et al.	Ophthalmic Plastic Surgery: Fundamentals	8 PERIODS M4, M-5, T-4, T-5 W-4, W-5, Th-4, Th-5
22	Iliff et al.	Ptosis	3 PERIODS W-4, W-5, W-6
23	Jardon & Laubheimer	Fitting Ocular Prostheses	2 PERIODS W-4, W-5
24	Knighton et al.	Practical Essentials of Glaucoma	9 PERIODS M-4, M-5, M-6 T-4, T-5, T-6 W-4, W-5, W-6

CONTINUOUS COURSES — OPHTHALMOLOGY

COURSE NO.	SPEAKER	SUBJECT	PERIODS
25	Leinfelder et al.	Diagnostic Aids in Neuro-ophthalmology	3 PERIODS Th-4, Th-5, Th-6
26	Lemoine et al.	Fundus Lesions	3 PERIODS M-4, M-5, M-6
27	Linksz	Physiology of Vision	2 PERIODS M-4, M-5
28	Lloyd	Diseases of the Cornea	2 PERIODS M-4, M-5
29	Masters, R., et al.	Slit Lamp Biomicroscopy: Fundamentals	9 PERIODS M-4, M-5, M-6 T-4, T-5, T-6 W-4, W-5, W-6
30	McLean et al.	.....SOLD OUT.....	
31	Mullen et al.	Ocular Emergencies	3 PERIODS M-4, M-5, M-6
32	Parks & Albert	Eso-Deviations	4 PERIODS W-5, W-6, Th-4, Th-5
33	Pierce et al.	Retinal Detachment: Basic Course	6 PERIODS M-4, M-5, T-4, T-5 W-4, W-5
34	Pischel et al.	Retinal Detachment	4 PERIODS M-4, M-5, T-4, T-5
35	Posner & Gorin	Office Gonioscopy	2 PERIODS T-4, T-5
36	Prewitt	Diagnosis & Treatment of Ocular Allergy	2 PERIODS W-5, W-6
37	Roberts	Glaucoma	2 PERIODS M-4, M-5
38	Rolett	Gonioscopy	2 PERIODS W-4, W-5
39	Romaine	Ocular Motility Surgery	4 PERIODS M-4, M-5, T-4, T-5
40	Romaine	Refraction Problems & Treatment	3 PERIODS M-6, T-6, W-6
41	Ruedemann & Noell	Clinical Electroretinography	2 PERIODS W-5, W-6
42	Schepens	.....SOLD OUT.....	
43	Schimek & Bousquet	Clinical Gonioscopy & Tonography	2 PERIODS Th-4, Th-5
44	Schlaegel & Hungerford	Uveitis	2 PERIODS Th-4, Th-5
45	Sloane	School Vision Testing	2 PERIODS M-4, M-5
46	Sloane	Telescopic Lenses	2 PERIODS T-4, T-5
47	Gettes	A. Prescription of Prisms B. Drugs in Refraction	2 PERIODS W-4, W-5
48	Stow & Moss	Diagnostic & Therapeutic Office Procedures	2 PERIODS M-5, M-6
49	Theobald et al.	Histopathology	12 PERIODS M-4, M-5, M-6, T-4 T-5, T-6, W-4, W-5 W-6, Th-4, Th-5, Th-6

CONTINUOUS COURSES — OPHTHALMOLOGY

COURSE NO.	SPEAKER	SUBJECT	PERIODS
50	Thorpe & Berman	Intraocular Foreign Bodies	2 PERIODS W-4, W-5
51	Thorpe et al.	.....SOLD OUT.....	
52	Thorpe & Allen, L.	Slit Lamp Gonioscopy	6 PERIODS M-4, M-5, M-6 T-4, T-5, T-6
53	Townes	Cataract Surgery	2 PERIODS Th-4, Th-5
54	Troutman et al.	Recent Advances in Ophthalmology	8 PERIODS M-5, M-6, T-5, T-6 W-5, W-6, Th-5, Th-6
55	Troutman	Enucleation Surgery	2 PERIODS T-5, T-6
56	Wise	Retinal Diseases	2 PERIODS Th-4, Th-5
57	Home Study Discussion Periods		10 PERIODS M-4, M-5, M-6, T-4 T-5, T-6, W-4, W-5 W-6, Th-4

SCHEDULE OF INDIVIDUAL COURSES—OPHTHALMOLOGY—Monday

Key M-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
104	Alvis	Glaucoma Surgery
123	Calhoun	Epithelization of the Anterior Chamber
126	Cassady	Toxoplasmic Retinochoroiditis
129	Chamberlain	Surgery of the Ocular Muscles
145	DeVoe	Surgery of Trauma
147	Diskan	Eye Screening in Schools
155	Everett	Retinal Detachment
158	Fernandez	Beta Radiation in Pterygium Surgery
163	Frayer	Retained Lens Protein After Operation & Trauma
198	Leopold & Apt	Chemotherapy
206	MacLean	Cataracts
227	Pfeiffer & Jones	Roentgenography of Eye & Orbit
233	Regan, E.	Cataract Surgery
241	Scarney	Slit Lamp Biomicroscopy
262	Veirs	External Dacryocystorhinostomy & Corrective Proceduces for Epiphora

Key M-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
102	Aldrich	Emotional Problems
109	Bahn	Orbital Disease
133	Cleasby	Iris Tumors
144	Day	Medical Exophthalmos
148	Duke	Lesions of the Lids
149	Dunlap	Divergence Anomalies
154	Ellis	Removal of Intraocular Foreign Bodies
159	Ferrer	Contact Lenses
169	Gipner	Recent Advances in Ophthalmic Surgery
237	Rundles	Streak Retinoscopy
244	Schlossman	Pleoptics
245	Schmidt	Applanation Tonometry
248	Simpson	Hemorrhage into the Vitreous
253	Spaeth, P., & Carmichael	Ocular Tumors

Key M-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
103	Allen, H.	Aseptic Technique in Ophthalmology
113	Berens	Cyclodiathermy, Cycloelectrolysis, Iridencleisis & Cyclodialysis
122	Buxton	Total & Partial Keratectomies
124	Carroll	Optic Nerve Lesions
131	Cholst	Central Fields
136	Coston	Inflammatory & Degenerative Disease of Retina
138	Cunningham	Types of Head Pain
141	Daily	Biomicroscopy of Vitreous & Fundus Oculi
160	Fink, W.	Binocular Problems in Routine Refraction
168	Gifford	Local Anesthesia with Special Reference to Deep Motor Block
170	Haik	Mechanics of Intracapsular Extraction
173	Henderson	Intracranial Arterial Aneurysms
175	Hunt	Problems in Accommodation
176	Jacobson	Clinical Electroretinography
190	Kornzweig	Clinical Embryology
193	Kronfeld	Medical Management of Eye Lesions Resulting in Increased Pressure
207	Mangold	Proper Fitting of Glasses
226	Perera	Retinal Detachment
238	Rychener	The Lacrimal Apparatus
242	Scheie	Congenital Glaucoma
257	Theodore	Ocular Therapeutics
264	Weisser	Repair of Corneal Injuries
265	Wendland	Magnetic Intraocular Foreign Bodies

SCHEDULE OF INDIVIDUAL COURSES—OPHTHALMOLOGY—Tuesday

Key T-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
105	Andrew	Eye Diseases in Children
108	Atkinson	Anesthesia
114	Berens	Ophthalmic Surgery
121-A	Buessler	Ophthalmic Radiology
123	Calhoun	Epithelization of the Anterior Chamber
125	Cassady	Dacryocystitis & Lacrimal Drainage
140	Cutler	Cataract
153	Egan	Cross Cylinder
164	Fread	Ideal Refraction Equipment
195	Leinfelder	Perimetry & Use of the Tangent Screen
199	Lepard	Eye Diseases Common to Children
209	Mann, W.	Interpretation of Functional Changes in Vision
218	Naquin	Dislocation of the Lens
236	Ruedemann	The Differential Diagnosis & Surgical Correction of Vertical Muscle Defects
250	Smith, J. L.	Neuro-ophthalmology
252	Spaeth, E.	Complicated Ptosis
266	Wood	Macular Lesions
267	Young & Dixon	Contact Lenses

Key T-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
111	Bannon	Aniseikonia
115	Bettman	Value of Goniotomy
128	Chamberlain	Vertical & Horizontal Components of Strabismus
143	Davis	Sutures in Cataract Surgery
149	Dunlap	Divergence Anomalies
157	Falls	Clinical Manifestations of Constitutional Syndromes Involving Head & Neck
166	Gartner	Fundus Diseases
186	Kennedy	Ocular Manifestations of Systemic Diseases
191	Kronenberg	Practical Ocular Therapeutics
197	Leopold	.....SOLD OUT.....
224	Paton	Corneal Transplantation
228	Pico	Dacryocystorhinostomy
243	Scheie	Penetrating Injuries of the Globe
249	Smith, B.	Fractures of the Orbit
260	Uihlein	Neuralgias of the Head

Key T-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
134	Coles	Office Management of Uveitis
141	Daily	Biomicroscopy of Vitreous & Fundus Oculi
146	DeVoe	Acute Surgical Lesions
161	Fink, W.	Vertical Muscle Surgery
165	Fry	Inflammatory & Degenerative Lesions at Posterior Pole of Eye
167	Gemeroy	External Diseases
169	Gipner	Recent Advances in Ophthalmic Surgery
180	Johnson, C. C.	Ptosis
189	Klien	Clinical Pathology of the Retina
200	Levy	Practical Uses of Contact Lenses
204	Long	Retinoblastoma & the Pseudoretinoblastomas
205	Lubkin	Psychosomatic Aspects of Ophthalmology
212	McCannel	Efficiency in the Office
213	McGraw	Contact Lenses
215	Merriam	Radiotherapy in Ophthalmology
216	Morrison	Glaucoma
232	Reese	Cataract Extraction
239	Salleras	Corneal Surgery
240	Sarrail	Plastic Lid Surgery
241	Scarney	Slit Lamp Biomicroscopy
246	Shipman	Scleral Resection
254	Sutliff	Practical Fitting of Corneal Lenses
259	Truhlsen	Home Orthoptics

SCHEDULE OF INDIVIDUAL COURSES—OPHTHALMOLOGY—Wednesday

Key W-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
101	**Aebli**	End Results in Muscle Surgery
106	**Asbury**	Practical Genetics in Everyday Office Practice
120	**Breakey**	Rehabilitation in Ophthalmology
121	**Breinin**	Electromyography
125	**Cassady**	Dacryocystitis & Lacrimal Drainage
127	**Castroviejo**	Corneal Surgery
139	**Cusick**	Ptosis Surgery
145	**DeVoe**	Surgery of Trauma
150	**Dunlap**	Accommodative Esotropia
162	**Fox**	Senile Ectropion & Entropion
172	**Hedges**	Neralgias of the Head
177	**Jaeckle**	Nonstrabismic Abnormalities of Binocular Vision
179	**Jobe**	Ophthalmic Instruments
185	**Kearns**	Unusual Neurologic Motor Anomalies
187	**King**	Lamellar Corneal Transplantation
195	**Leinfelder**	Perimetry & Use of the Tangent Screen
203	**Lloyd**	Fundus Lesions of the Senile Eye
208	**Mann, M.**	Vascular Changes in the Fundus
242	**Scheie**	Congenital Glaucoma
251	**Spaeth, E.**	Plastic Surgery
263	**Weingeist**	Lacrimal Sac Surgery

Key W-5 — 3:15 p.m. to 4:15 p.m.

Course No.	SPEAKER	SUBJECT
110	**Bannon**	Practical Aspects of Multifocal Corrections
112	**Belmont**	Refraction
119	**Brav**	Diseases of the Choroid
130	**Chamlin**	Perimetry
151	**Dunnington**	Cataract Extraction
156	**Falls**	Ocular Geriatrics
183	**Katz, D.**	Ophthalmic Anatomy
194	**Leahey**	Corneal Transplantation
196	**Lennon**	Angle Closure Glaucoma
213	**McGraw**	Contact Lenses
214	**McGuire**	Cataract
225	**Paulson**	Lighting for Comfortable & Accurate Seeing
229	**Pischel**	Problems with Light Therapy
231	**Potts**	Retinotoxic Substances
234	**Rizzuti**	Tumors of Lids & Ocular Adnexa

Key W-6 — 4:30 p.m. to 5:30 p.m.

Course No.	SPEAKER	SUBJECT
107	**Ascher**	Cyclodialysis
116	**Bettman**	Radioactive Isotopes
135	**Cordes**	Congenital Cataract
171	**Halberg**	Tear Formation
178	**Jampolsky**	Ocular Divergence
181	**Joy**	Chronic Glaucoma
182	**Kasper**	Dacryocystorhinostomy
184	**Katz, I.**	Common Eye Diseases of Dogs & Their Therapy
191	**Kronenberg**	Practical Ocular Therapeutics
192	**Kronfeld**	Present Trends in Ocular Pharmacology
201	**Lewis**	Practical Use of Mechanical Visual Aids in the Poor Sighted
211	**Marshall**	Differential Diagnosis & Treatment of Infections of Lids
217	**Murrah**	Subnormal Vision
219	**Newell**	Radioactive Isotopes
221	**Ogle**	Stereopsis
235	**Rubin**	Aims in Management of Squint
247	**Simonton**	Orbital Surgery
255	**Tait**	Heterophoric Asthenopia
256	**Tassman**	Eye Involvement in Systemic Diseases
258	**Trent**	Progress in Ophthalmology
260	**Uihlein**	Neuralgias of the Head
261	**Vail**	Senile Cataract

SCHEDULE OF INDIVIDUAL COURSES—OPHTHALMOLOGY—Thursday

Key Th-4 — 2:00 p.m. to 3:00 p.m.

Course No.	SPEAKER	SUBJECT
117	Block	Office Practice of Ophthalmology
118	Bonaccolto	Dacryocystorhinostomy
132	Clark, W.	Surgery of Ocular Muscles
137	Cowen	Pool Gonioscopy
142	Davidson	Surgical Instruments
152	Edwards	Abnormal Effects of Prescribed Lenses on Extrinsic Muscles
174	Hill, H.	Cataract Surgery
188	King	Transplantation of Preserved Ocular Tissues
202	Linn	Evaluation of Patient for Surgery in Open Angle Glaucoma
210	Marks	Electrical Currents Used in Ophthalmology
220	Newell	Refinements in Ophthalmic Surgery
222	Ogle	Binocular Oculomotor Coordination & Imbalances
223	Olson	Iritis, Iridocyclitis, Keratitis
230	Post	Refinements of Refraction

SECTION ON INSTRUCTION

ADVANCE ORDER SHEET

All courses cost $2.00 for each period (one hour). Follow instructions explicitly and check for errors in listing before mailing.

Orders will not be accepted for presale after September 29. To ensure acceptance they should be mailed before September 27 to

WILLIAM L. BENEDICT, M.D., *Executive Secretary-Treasurer*
American Academy of Ophthalmology and Otolaryngology
15 Second Street S.W., Rochester, Minnesota

Academy Candidates and Guests May Purchase Tickets ONLY at Instruction Desk in Registration Foyer

(Over)

ADVANCE ORDER SHEET

ORDERS FILLED ONLY IF CURRENT DUES ARE PAID

NO REFUNDS WILL BE MADE AFTER OCTOBER 7

(Type or Print) Last Name First Name Middle Initial

Office Address

City Zone State

Days to be Present

- ☐ Monday
- ☐ Tuesday
- ☐ Wednesday
- ☐ Thursday

Total Amount Enclosed $_____

SELECTION OF COURSES ON REVERSE SIDE OF THIS BLANK

RECAPITULATION OF GENERAL INSTRUCTIONS

1. Monday, Tuesday, Wednesday, and Thursday are represented by the letters M, T, W, Th. The first period in the morning is represented by figure 1, second period by figure 2 and third period by figure 3. The first period in the afternoon is represented by figure 4, second period by figure 5, and third period by figure 6. All courses in otolaryngology and maxillofacial surgery are presented in the mornings. All courses in ophthalmology are presented in the afternoons.
2. Reservation of instruction tickets in advance of the meeting will be made ONLY for MEMBERS whose 1960 dues have been paid. ACADEMY CANDIDATES AND GUESTS MAY MAKE SELECTION OF COURSES *ONLY* AT THE GENERAL REGISTRATION DESK IN CHICAGO.
3. Make check payable to W. L. Benedict, M.D.
4. Mail order promptly to ensure reservation of first choices. If necessary, an attempt will be made to rearrange schedule to include "first choices."

(Over)

SELECTION OF COURSES

PERIOD		FIRST CHOICE		SECOND CHOICE		THIRD CHOICE	
		Course No.	Lecturer	Course No.	Lecturer	Course No.	Lecturer
MON.	4						
P.M.	5						
	6						
TUES.	1						
A.M.	2						
	3						
P.M.	4						
	5						
	6						
WED.	1						
A.M.	2						
	3						
P.M.	4						
	5						
	6						
THURS.	1						
A.M.	2						
	3						
P.M.	4						
	5						
	6						

CONTINUOUS COURSES AND PANEL DISCUSSIONS
OTOLARYNGOLOGY

COURSE NO.	SPEAKER	SUBJECT	PERIODS
301	Anderson	Rhinoplasty	2 PERIODS T-1, T-2
302	Baron	Tympanoplastic Procedures	2 PERIODS Th-1, Th-2
303	Brewer & Briess	Head & Neck Symptoms Associated with Laryngeal Microtrauma	2 PERIODS Th-1, Th-2
304 or 305	Conley & Lenz	Carcinoma of Head & Neck	2 PERIODS T-1, T-2 or W-1, W-2
306	Converse	Principles of Nasal Plastic Surgery—Basic	2 PERIODS T-2, T-3
307	Converse	Principles of Nasal Plastic Surgery—Advanced	2 PERIODS Th-2, Th-3
308	Fox & Glorig	Noise in Industry	2 PERIODS T-2, T-3
309	Godlowski	I. Physiopathology of Endocrine System II. Enzymatic Mechanism of Allergy	2 PERIODS T-2, T-3
310	Godlowski	Endocrinopathies	2 PERIODS Th-2, Th-3
311	Guilford & Wright	Techniques of Tympanoplasty	2 PERIODS W-1, W-2
312	Hansel	Allergy & Medical Otolaryngology	2 PERIODS T-2, T-3
313	Harbert	Clinical Audiology	2 PERIODS T-1, T-2
314	Hardy & Pauls	Clinical Audiology for Children	2 PERIODS W-2, W-3
315	House, H., & House, W.	Reconstructive Surgery for Chronic Ear Disease	2 PERIODS W-1, W-2
316	Johnston et al.	Emergencies in Otolaryngology	2 PERIODS W-1, W-2
317	Kazanjian et al.	Traumatic Injuries of the Face	2 PERIODS W-2, W-3
318	Lathrop et al.	Your Otological Problems	2 PERIODS Th-2, Th-3
319	Lawrence	Acoustic Principles in Middle Ear Surgery	2 PERIODS W-2, W-3
320 or 321	Lindsay	Deafness & Dizziness of Vascular Origin	2 PERIODS T-2, T-3 or W-2, W-3
322	Simonton et al.	What the Otologist Should Know About Hearing Aids	2 PERIODS Th-2, Th-3
323	Sisson et al.	Team Judgment in the Management of Head & Neck Cancer	2 PERIODS T-2, T-3
324	Walsh	What Can Be Done for Deafness	2 PERIODS Th-1, Th-2

SCHEDULE OF INDIVIDUAL COURSES—OTOLARYNGOLOGY—Tuesday

Key T-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
401	Alexander	The Adenoid Problem
412	Bordley	Deafness in the Infant and Preschool Child
418	Compere	Radiologic Examination of Mastoid, Middle Ear & Eustachian Tube
420	Daly & Cardona	Corrosive Esophagitis
422	DeWeese	The Problem of the Dizzy Patient
425	Farrior, J. B.	Tympanoplasty in 3-D
429	Fishbein	Medical Writing
433	Furstenberg	Management of Nasal Obstruction
437	Goodhill & Brockman	The Articulated Prosthesis in Stapes Surgery
441	Guilford & Wright	Controlled Healing of Tympanoplasty & Mastoid Cavities
447	Hilsinger	Vascular Headaches
458	Kos	.....SOLD OUT.....
466	Loch	Septum Deviations
467	Lore	Vascular Surgery in Head & Neck
470	McHugh	Traumatic Conductive Deafness & Facial Paralysis
471	McLaurin & Raggio	The Child Without Speech
507	Tabb	.....SOLD OUT.....
508	Torok	Clinical Nystagmography
511	Wallner	Diseases of the Larynx

Key T-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
426	Farrior, J. B.	Stapes Operations—1960
431	Fitz-Hugh & McLean	Cancer of the Nasal Accessory Sinuses
432	Fowler	Facial Paralysis
438	Gorney	Septum Surgery in the Twisted Nose
443	Hallberg	Severe Nosebleed
453	Jordan, L.	Recent Nasal Fractures
462	Lepper	Antibiotics in Otolaryngology
478	Pastore	The Throat: Nonsurgical Problems
479	Pearlman, S.	Certain Aspects of Aging in Ear, Nose & Throat Regions
487	Reed	Neck Dissection
501	Shambaugh	Surgical Application of the Mechanics of the Middle Ear
502	Shea	Fenestration of the Oval Window
510	von Leden & Moore	Current Trends in Office Laryngology

Key T-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
403	Altmann & Waltner	Treatment of Meniere's Disease with Ultrasonic Waves
404	Anson	Stapediovestibular Anatomy in Relation to the Problems of Stapes Surgery
411	Bolotow	Rhinoplasty
421	Devine	Total Laryngectomy
439	Grandon	Plastic Surgical Procedures
445	Harkins	Routine Office Management of Allergic Patients
454	Jordan, R.	Meniere's Disease
455	Juers	Perforations & Hearing Conservation Surgery
461	Lemon	Serous Otitis Media in Children
473	Myers	The Microsurgery of Otosclerosis
475	Neivert	Intranasal Ethmoidectomy
482	Proctor	Tympanoplasty
489	Rinkel	The Diagnosis of Food Allergy
496	Schuknecht	.....SOLD OUT.....

SCHEDULE OF INDIVIDUAL COURSES—OTOLARYNGOLOGY—Wednesday

Key W-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
402	Alfaro	Fullness in the Ear
405	Armstrong	Use of Vinyl Tubing in the Treatment of Chronic Secretory Otitis Media
413	Bradley	Practical Office Speech Audiometry
415	Ciell	Hypnosis in Otolaryngology
422	DeWeese	The Problem of the Dizzy Patient
427	Farrior, R.	A Method of Otoplasty
435	Goldman, I.	Correction of the Deviated Nose
436	Goldman, J.	Nasal Disorders
452	Jones	External Otitis
458	Kos	Vein Plug Stapedioplasty
474	Myers & Schlosser	Surgery of Chronic Otitis Media & Mastoiditis
486	Rawlins	The Corticosteroids in Otolaryngology
488	Reger	Practical Office Audiometry
498	Sellers	The Office Practice of Otolaryngology
506	Sullivan et al.	Facial Palsies & Their Treatment
507	Tabb	Closure of Tympanic Membrane Perforations with Vein Grafts
512	Wallner	Diseases of the Nasopharynx

Key W-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
410	Bellucci	Surgery of the Stapes & Oval Window
414	Brodnitz	Actor & Singer as Patients of the Otolaryngologist
444	Hallberg	Sudden Deafness
457	Kirchner	Facial Bone Injuries
459	Kraus & Furey	Adventures in Space
464	Lewis, P.	Reactions to Drugs & Antibiotics
465	Lewy	Direct Laryngoscopy
468	Magielski	Neurogenic Tumors of Head & Neck
480	Perlman, H.	Fluid in the Middle Ear
491	Ronis	Chronic Sinusitis
495	Scheldrup	Applied Anatomy of the Neck
500	Senturia & Adler	Middle Ear Effusions
503	Skolnik	Electrodiagnosis in Facial Nerve Paralysis
513	Woodman	Vocal Cord Surgery

Key W-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
404	Anson	Stapediovestibular Anatomy in Relation to the Problems of Stapes Surgery
406	Arnold	Radiographic Diagnosis of Temporal Bone Lesions
409	Bell	Correction of Outstanding Ears
416	Cocke	Salivary Gland Tumors
417	Cody	Eustachian Tube
419	Corgill	Radical Hypopharyngeal Surgery Combined with Neck Dissection
424	Erich	Fractures of the Facial Bones
430	Fitz-Hugh & McLean	Salivary Glands
440	Guggenheim	Technique & Pitfalls of Adenoidectomy
455	Juers	Perforations & Hearing Conservation Surgery
456	Kemler	Diagnostic & Therapeutic Aids in ENT Practice
460	Lederer	Diagnostic & Therapeutic Pitfalls in ENT Practice
469	McBean	Secretory Otitis Media in Children
476	Ogura & Som	Conservation of Laryngeal Function in Cancer of the Larynx
481	Petryshyn	The Fractured Mandible
482	Proctor	Tympanoplasty
502	Shea	Fenestration of the Oval Window

SCHEDULE OF INDIVIDUAL COURSES—OTOLARYNGOLOGY—Thursday

Key Th-1 — 9:00 a.m. to 10:00 a.m.

Course No.	SPEAKER	SUBJECT
402	Alfaro	Fullness in the Ear
407	Atkins	Bronchoscopic Problems of Infancy & Childhood
415	Ciell	Hypnosis in Otolaryngology
424	Erich	Fractures of the Facial Bones
435	Goldman, I.	Correction of the Deviated Nose
437	Goodhill & Brockman	The Articulated Prosthesis in Stapes Surgery
441	Guilford & Wright	Controlled Healing of Tympanoplasty & Mastoid Cavities
448	Hough	Anatomic Variations in the Middle Ear
452	Jones	External Otitis
466	Loch	Septum Deviations
472	Missal	The Cervico-occipital Syndrome
484	Proud	Middle Ear Effusion
492	Rosen	Surgical Anatomy of the Vestibule with Reference to the Stapedial Footplate
493	Ryan	Headache
497	Schuknecht	Removal & Preparation of the Human Temporal Bone

Key Th-2 — 10:15 a.m. to 11:15 a.m.

Course No.	SPEAKER	SUBJECT
408	Becker	Plastic Surgery of Protruding Ears
410	Bellucci	Surgery of the Stapes & Oval Window
414-A	Carhart	Auditory Tests in Diagnosis
423	Dickinson	Saddle Nose
434	Glorig	The Otolaryngologist & Hearing Aid Selection
446	Hilger	Office Diagnostic Methods & Treatments
463	Lewis, J.	Surgery for Advanced Ear Cancer
483	Proctor	Surgical Anatomy of the Temporal Bone
494	Saunders	Oral Lesions
499	Sellers	Dizziness
509	Van Alyea	Sinusitis

Key Th-3 — 11:30 a.m. to 12:30 p.m.

Course No.	SPEAKER	SUBJECT
409	Bell	Correction of Outstanding Ears
428	Farrior, R.	Cancer of Head & Neck
442	Guilford & Wright	Stapes Mobilization Techniques
449	House, H.	Stapes Mobilization Procedure
451	Johnson, F.	Tonsillectomy & Adenoidectomy
460	Lederer	Diagnostic & Therapeutic Pitfalls in ENT Practice
469	McBean	Secretory Otitis Media in Children
477	Ogura	Radical Neck Dissection & Laryngeal Carcinoma
485	Rabbett	Submucous Resection of the Nasal Septum
490	Rinkel	The Diagnosis of Inhalant Allergy
504	Skolnik	Head & Neck Surgery
505	Spector	Dizziness

LISTING OF AUDIOMETERS

The following audiometers are recommended for listing by a subcommittee of the Committee on Conservation of Hearing, American Academy of Ophthalmology and Otolaryngology:

PURE TONE AUDIOMETERS

Beltone Model 10-C Model 15-C	Beltone Hearing Aid Company Audiometer Division 2900 West 36th Street Chicago 32, Illinois
Maico Model MA-5 Model H1-B for pure tones only	Maico Electronics Inc. Maico Building 21 North Third Street Minneapolis 1, Minnesota
Sonotone Model 91	Sonotone Corporation Elmsford, New York

SPEECH AUDIOMETERS

Grayson-Stadler Model 162	Grayson-Stadler Co. West Concord, Massachusetts

SCREENING AUDIOMETERS

Ambco Otometer 600	Ambco Inc. 1222 W. Washington Blvd. Los Angeles 7, California

Each model number in the list as a rule indicates the most advanced model of that series. If less advanced instruments are for sale under similar model numbers, it is assumed that they will function as well or better than the more complicated model in a series.

Acceptance for listing means that the audiometer manufacturer has demonstrated the ability to produce one instrument that performs according to A.S.A. specification. It does not imply that any individual instrument will perform within the tolerances. It is obviously beyond the scope of the Listing Committee or the Referee Laboratories to provide production control for audiometer manufacturers and their customers; however, listing does indicate that the product of the manufacturer can come up to 1951 A.S.A. specifications, and it is up to the customer to carry on negotiation with the manufacturer if a particular instrument which he receives is faulty.

Abatron D-100 and Ambco Oto-chek 800 were removed from the above list at the request of the respective manufacturers.

NEWS AND ANNOUNCEMENTS

PROFESSOR TUTOMU SATO

We have learned with regret of the death of Professor Tutomu Sato, the head of the Department of Ophthalmology, Juntendo University, Tokyo, Japan. He died June 9, 1960, of a heart attack.

FALL MEETINGS

The Iowa Academy of Ophthalmology and Otolaryngology

The Iowa Academy of Ophthalmology and Otolaryngology will meet September 14 to 16 at The New Inn, Okoboji, Iowa.

Speakers will include Robert H. Foss, M.D., Des Moines; Mrs. Ruby Hopkins, New York; Chester C. Woodburn, Jr., M.D., Des Moines; John E. Harris, M.D., Minneapolis, and Jerome A. Hilger, M.D., St. Paul.

Nonmember physicians interested in Ophthalmology and Otolaryngology are welcome. There will be a registration fee of $10.00 for all physicians not members of the Iowa Academy.

American Society for Head and Neck Surgery

Dr. W. Franklin Keim, secretary for American Society for Head and Neck Surgery, has announced that the Society will hold a meeting in the Crystal Room of the Palmer House on Sunday evening, October 9, at eight o'clock. All interested persons are invited to attend.

Montreal Ophthalmological Society

The Montreal Ophthalmological Society will meet at the Royal Victoria Hospital, Montreal, Wednesday evening, November 2, 1960. The guest speaker will be Dr. Gordon M. Bruce, Professor of Clinical Ophthalmology, College of Physicians and Surgeons, Columbia University, New York. The title of his address is "The A-V Syndromes." Papers by Society members will complete the program.

POSTGRADUATE COURSES

Temple University

The Department of Laryngology and Bronchoesophagology, Temple University Medical Center, will sponsor two postgraduate courses under the direction of Drs. Chevalier L. Jackson and Charles M. Norris.

Laryngology and Laryngeal Surgery will be given from September 26 to October 7, 1960, and from April 10 to 21, 1961. Bronchoesophagology will be given November 7 to 18, 1960, and from February 6 to 17, 1961.

The tuition fee for each course is $250. Application and further information may be obtained by writing Jackson-Research, Temple University Medical School, 3400 N. Broad St., Philadelphia 40.

Temple University

A "Short Course in Audiology" is offered several times each year by the Section of Audiology, Department of Otorhinology, Temple University School of Medicine, Philadelphia 40.

The course is one week in length and encompasses theoretical and clinical aspects of audition. Hearing testing and the interpretation of the results are stressed. Office procedures, equipment and industrial audiometry are discussed.

Further information may be obtained by writing to Philip E. Rosenberg, Ph.D., Director, Section of Audiology, Temple University Medical Center, 3401 North Broad St., Philadelphia.

Emory University

A postgraduate course in "Techniques in Ophthalmic Surgery" will be presented on December 1 and 2, 1960, at the Grady Memorial Hospital, Atlanta, Ga., under the sponsorship of the Grady Clay Memorial Eye Clinic and the Department of Ophthalmology of the Emory University School of Medicine.

The course is designed for the practicing clinical ophthalmologist and will consist of lectures and panel discussions by Dr. Frank Costenbader, Chairman, Department of Ophthalmology, Children's Hospital, Washington, D. C.; Dr. John McLean, Professor of Ophthalmology, Cornell University School of Medicine, New York; and Dr. Harold Scheie, Professor of Ophthalmology, University of Pennsylvania School of Medicine, Philadelphia.

Diagnostic principles and techniques, preoperative and postoperative management, surgical principles and techniques in extraocular muscle surgery, cataract surgery, and glaucoma surgery will be discussed by these distinguished surgeons.

THE NATIONAL COUNCIL TO COMBAT BLINDNESS

The National Council to Combat Blindness, Inc., has announced that the closing date for receipt of completed applications for FIGHT FOR SIGHT full-time research fellowships, grants-in-aid and summer student fellowships for the 1961-1962 period has been designated as March 1, 1961.

In general, applicants for full-time research fellowships and grants-in-aid will be notified in July, with the first of August as the commencement date for the project. Under special circumstances, where earlier notification is essential, the Scientific Advisory Committee may consider applications in advance of the scheduled date.

Applicants for student fellowships will be notified in May of the action taken by the Scientific Advisory Committee in order that arrangements may be made with their respective institutions to commence work in early summer.

Appropriate forms may be obtained by addressing Secretary, National Council to Combat Blindness, Inc., 41 West 57th St., New York 19.

The National Council to Combat Blindness, Inc., was founded in 1946 with the primary purpose of financing research in ophthalmology and related sciences. The objective of its program is the ultimate reduction of blinding eye diseases and ocular disorders, through increased basic and clinical research in the field of scientific investigation.

Grants and fellowships approved at the June 1960 meeting of the organization's Scientific Advisory Committee will be announced as soon as all official acceptances have been received.

Goodwin M. Breinin, M.D., Chairman, Department of Ophthalmology, New York University-Bellevue Medical Center, and Harold G. Scheie, M.D., Chairman, Department of Ophthalmology, University of Pennsylvania, School of Medicine, have recently joined the Advisory Committee.

POSITIONS

POSITIONS AVAILABLE

WANTED: YOUNG OTOLARYNGOLOGIST, recently trained and Board-qualified, to join 12-man group in city of 30,000. Beginning salary $20,000 per year. For further information, write P. W. Malone, M.D., Malone and Hogan Clinic, 811 Main, Big Spring, Texas.

WANTED: OPHTHALMOLOGIST OR E.E.N.T. MAN—For immediate occupancy, the best facilities and location for a well-qualified ophthalmologist or E.E.N.T. man. Private practice in Lake Geneva, Wisconsin. City noted for its excellent schools, churches, and varied sports. For further information, write E. D. Hudson, M.D., Hudson Clinic, Lake Geneva, Wisconsin, or call Chestnut 8-6232, Lake Geneva.

WANTED: ENT OR EENT SPECIALIST to take over Eye, Ear, Nose and Throat practice of deceased doctor. Completely equipped office in same building 30 years. Immediately available. For further information, write Charles E. Parrish, executor, 528 Medical Arts Building, San Antonio, Texas.

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WANTED—Location for private practice of OPHTHALMOLOGY in California or Missouri. Prefer small or medium-sized town.

Board-certified OPHTHALMOLOGIST, 31 years old, member of American Medical Association, Minnesota State Medical Society, Jules Gonin Club, and Society of Military Ophthalmologists. B.S. degree from Northwestern University, 1950; M.D. degree from Cornell University, 1954; internship at Barnes Hospital (St. Louis, Mo.), 1954-1955; eye residency at Mayo Clinic, 1955-1958; United States Army, 1958-1960.

Available in December 1960. For further information, write Richard K. Lansche, Captain, MC, USA, 11th Field Hospital, APO 178, U. S. Forces, New York, N. Y. (After October 12, write Dr. Richard K. Lansche, 8358 Racquet Drive, St. Louis 21, Mo.)

Listings of positions available should be sent to W. L. Benedict, M.D., 15 Second Street S. W., Rochester, Minn., by the fifth of the month preceding publication. They should include (1) type of physician wanted (ophthalmologist or otolaryngologist: diplomate, resident, etc.) (2) type of position to be filled; and (3) to whom to write for further information. Unless otherwise requested, listings will be published only once.

The Academy will handle no further correspondence beyond the listing and assume no responsibility. Neither does it endorse nor guarantee any of the published listings.

TEACHING AIDS

MANUALS

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EMBRYOLOGY OF THE HEAD AND NECK, Jack Davies	1.00
RADIOGRAPHIC ANATOMY OF THE TEMPORAL BONE, J. Brown Farrior	1.00
ALLERGY IN OTOLARYNGOLOGY, F. K. Hansel and J. R. Anderson	2.00
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NONSURGICAL COMPLICATIONS OF OTOLARYNGOLOGIC SURGERY, J. J. Pressman, et al.	1.00

MONOGRAPHS

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HEARING TESTS, Arthur L. Juers	0.25
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ABSTRACTS

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